

Utility of Near-infrared Spectroscopy Monitoring in the Management of Hyperperfusion Syndrome after Intracranial Carotid Artery Stenting

Shuntaro Togashi,^{1,2} Hiroaki Shimizu,² and Yoshitaka Suda¹

Cerebral hyperperfusion syndrome (HPS) is a rare complication of carotid endarterectomy (CEA) and stenting. There are only a few case reports about HPS after intracranial stenting, and its clinical course remains unclear. We report an unusual case of HPS after intracranial internal carotid artery (ICA) stenting. An 87-year-old woman underwent uneventful balloon angioplasty for the right intracranial ICA one year ago; then she presented with restenosis at the same arterial location. She then underwent an ICA stent placement procedure. Preoperative cerebral blood flow (CBF) studies revealed hemodynamic ischemia. At the time of surgery, the stenotic lesion was near-occlusion. Twelve hours after the successful stenting procedure, the patient became restless, and near-infrared spectroscopy (NIRS) indicated a blood flow increase to the affected side. Arterial spin labeling (ASL) imaging performed on the same day showed high signal intensity only in the right hemisphere. She was treated with sedation, blood pressure control, and minocycline hydrochloride administration. Because of the strict management under continuous monitoring with NIRS, her symptoms gradually improved over the next 6 days. The right-to-left difference observed with ASL imaging resolved 6 days after surgery, and she was discharged with no neurological deficit. This case highlights the utility of NIRS monitoring in the management of HPS after intracranial stenting.

Keywords: hyperperfusion syndrome, intracranial stenting, near-infrared spectroscopy

Introduction

Cerebral hyperperfusion syndrome (HPS) is a rare, but potentially fatal complication of carotid endarterectomy (CEA) and carotid artery stenting (CAS).^{1,2} Cerebral HPS occurs in about 1.9% after CEA and in about 1.2% after CAS.³ Although instances of HPS after both CEA and CAS have been well documented, only a few cases have been reported after endovascular treatment including intracranial stenting.^{4–9} Herein, we report an unusual case of HPS after

intracranial internal carotid artery (ICA) stenting that was monitored over time with near-infrared spectroscopy (NIRS).

Case Report

An 87-year-old woman initially presented with recurrent episodes of left upper motor weakness one year ago. Magnetic resonance imaging (MRI) demonstrated acute infarction in the right frontal and parietal lobes (Fig. 1A). Severe stenosis (70%, according to the Warfarin-Aspirin Symptomatic Intracranial Disease criteria¹⁰) with poor collateral flow was observed at the cavernous portion of the right ICA (Figs. 1B and 1C).

N-isopropyl-p-[¹²³I] iodoamphetamine single-photon emission computed tomography (SPECT) revealed normal cerebral blood flow (CBF) and a significant decrease in cerebrovascular reactivity (CVR) in the right hemisphere, which was classified as type 2 based on the Kuroda classification.¹¹

Balloon angioplasty performed under general anesthesia to prevent further ischemia was uneventful after the addition of dual antiplatelet therapy (DAPT, cilostazol 200 mg per day and clopidogrel 75 mg per day) and 1 month of rehabilitation. The internal diameter of the stenotic lesion site increased from 0.86 to 1.47 mm (Fig. 1D).

The postoperative course was uneventful, and she was discharged with no neurological deficit. Although she continued to be treated with single-antiplatelet therapy (cilostazol 200 mg per day), pitavastatin, and strict blood pressure control, follow-up angiography showed restenosis at the same location 9 months after the first operation (Fig. 2A).

N-isopropyl-p-[¹²³I] iodoamphetamine SPECT revealed a slight decrease in the resting CBF (87% of that of the contralateral hemisphere) and a significant decrease in CVR in the right hemisphere (1.8% vs. 53% in the contralateral hemisphere; Figs. 2B and 2C), suggestive of Kuroda type 3 ischemia.

After 2 weeks of DAPT, she underwent a stenting procedure under general anesthesia, 1 year after the first surgery. NIRS was used for intraoperative CBF monitoring. On the affected side, the sensor was attached to the middle cerebral artery territory at the temporal side because the right anterior cerebral artery (ACA) was hypoplastic. At the time of surgery, the stenotic lesion was near-occlusion (Fig. 3A). After intravenous administration of 6000 U heparin, the lesion was bypassed with a 0.014-inch guide wire and pre-dilated with a 3 mm × 9 mm Gateway percutaneous transluminal angioplasty balloon catheter (Boston Scientific, Marlborough, MA, USA). A 4 mm × 20 mm Wingspan stent was then deployed (Fig. 3B). The stenotic lesion was sufficiently expanded immediately after stent placement, and the ipsilateral value of NIRS demonstrated an increase from 60/66 (right side/left side) to 66/66

¹Department of Neurosurgery, Yuri Kumiai General Hospital, Yurijonjo, Akita, Japan

²Department of Neurosurgery, Akita University Graduate School of Medicine, Akita, Akita, Japan

Received: January 6, 2020; Accepted: March 24, 2020
Online September 17, 2020

Copyright© 2020 by The Japan Neurosurgical Society
This work is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives International License.

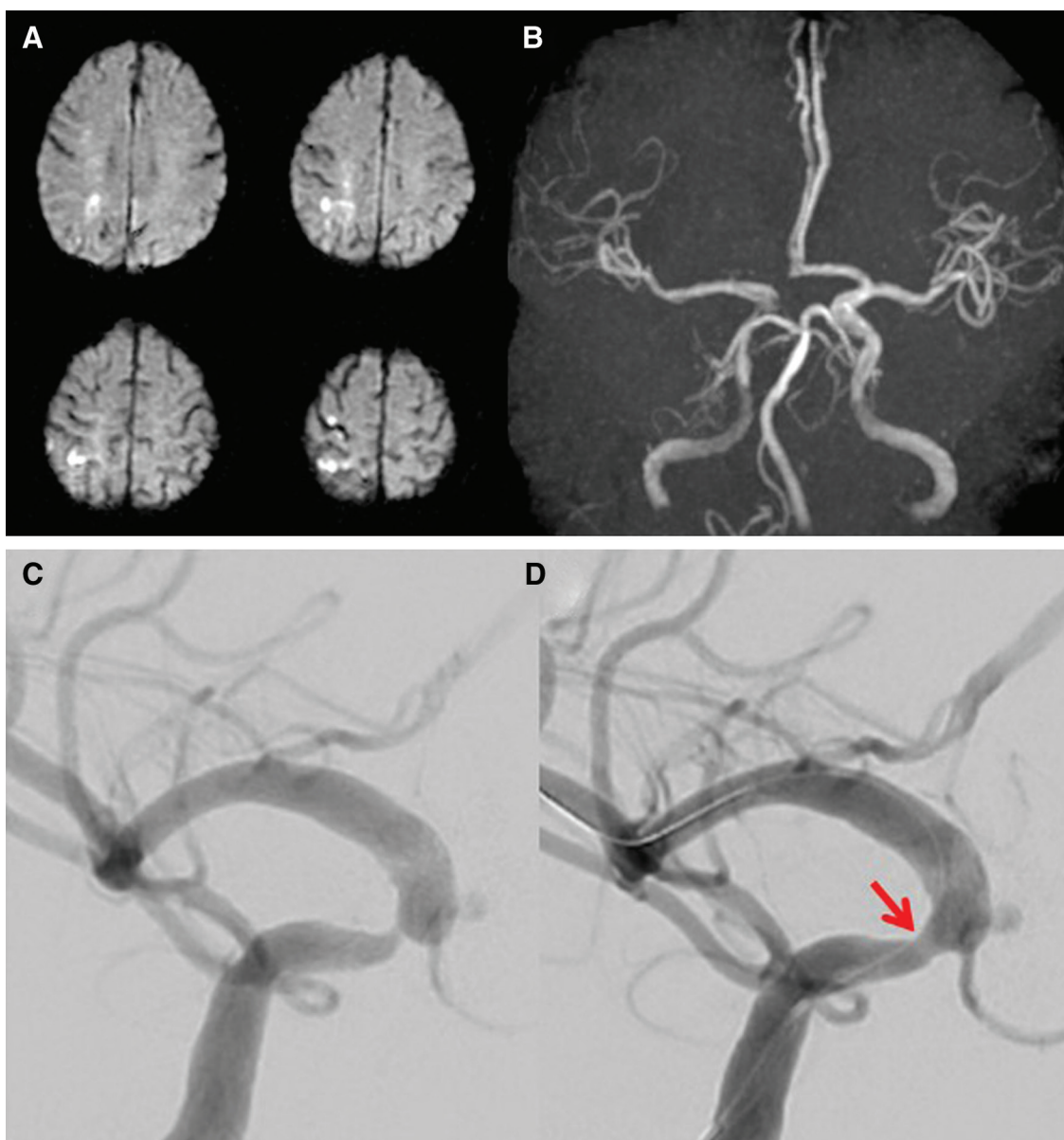


Fig. 1 Initial MRI and intraoperative findings of the balloon angioplasty. (A) Initial diffusion-weighted imaging showing infarction in the right hemisphere. (B) Initial magnetic resonance angiography showing low intensity of the right ICA angiogram, before (C), and after (D) the balloon angioplasty procedure in the first surgery, showing improvement in the stenosis (arrow). ICA: internal carotid artery; MRI: magnetic resonance imaging.

(Fig. 4A). Because the right-to-left difference in the NIRS increased by 8 points immediately after surgery, hyperperfusion was suspected and the patient was treated with propofol for sedation, nicardipine hydrochloride for blood pressure control, and minocycline hydrochloride for neuroprotection.¹²⁾

Although systolic blood pressure was controlled to 100–120 mmHg, she became restless 24 hours after the surgery, with a 17-point increase in the right-to-left difference in the NIRS (70/53) (Fig. 4B). Pulsed continuous arterial spin labeling (ASL) imaging with a post-labeling delay of 1.5 ms (Optima MR450w 1.5T, GE Healthcare Japan Ltd, Hino, Tokyo, Japan) showed a high signal intensity in the right hemisphere compared to the contralateral side (Fig. 4C), consistent with HPS.

Strict management under continuous monitoring with NIRS was performed. After 6 days of sedation, NIRS improved to 53/50 (Fig. 4D), and ASL imaging revealed no right-to-left difference (Fig. 4E). The sedation was discontinued on the same day. N-isopropyl-p-[¹²³I] iodoamphetamine SPECT, 20 days after the operation, demonstrated improvement in the CBF in the right hemisphere (Fig. 5). Her consciousness level gradually improved over the next few days, and she was discharged with no neurological deficit after 1 month of rehabilitation.

Discussion

Only a few case reports have documented the incidence of HPS after endovascular treatment including intracranial

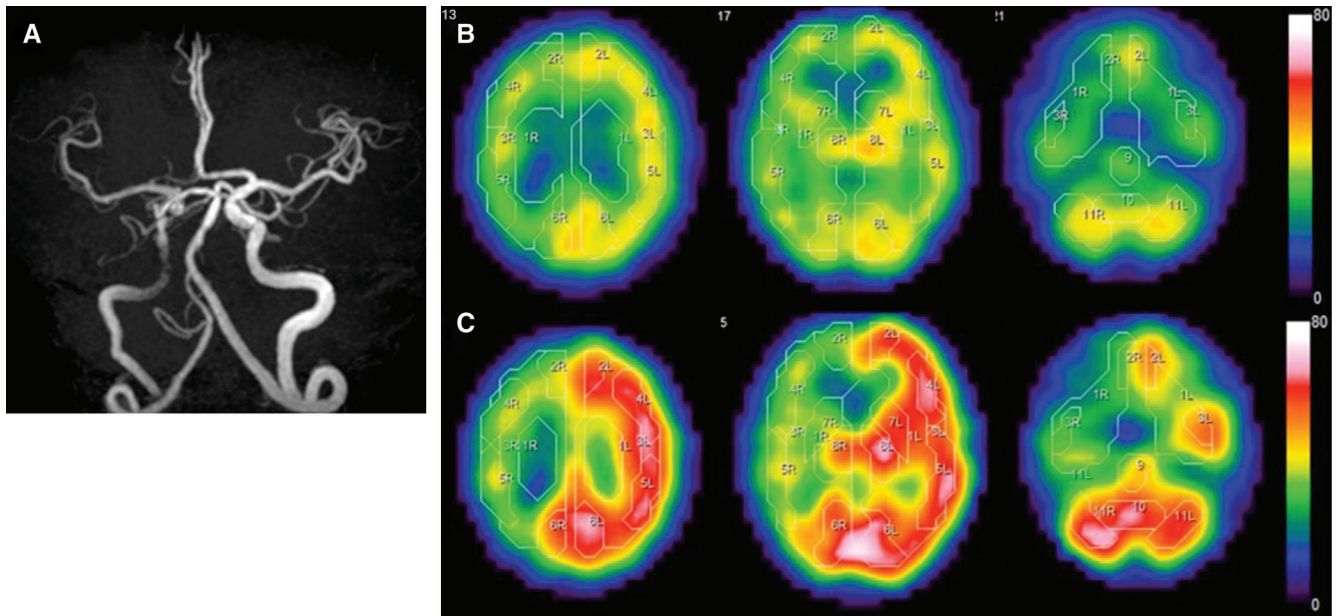


Fig. 2 Findings of magnetic resonance angiography and SPECT before intracranial stenting. (A) Magnetic resonance angiography performed 9 months after the first surgery, showing low intensity of the right ICA. (B) N-isopropyl-p-[¹²³I] iodoamphetamine SPECT performed before the second surgery demonstrating a slight decrease in the right hemispheric CBF at rest (87% of that of the contralateral hemisphere) and (C), demonstrating a significant decrease in CVR after administration of acetazolamide (1.8% vs. 53% in the contralateral hemisphere). CBF: cerebral blood flow; CVR: cerebrovascular reactivity; ICA: internal carotid artery; SPECT: single-photon emission computed tomography.

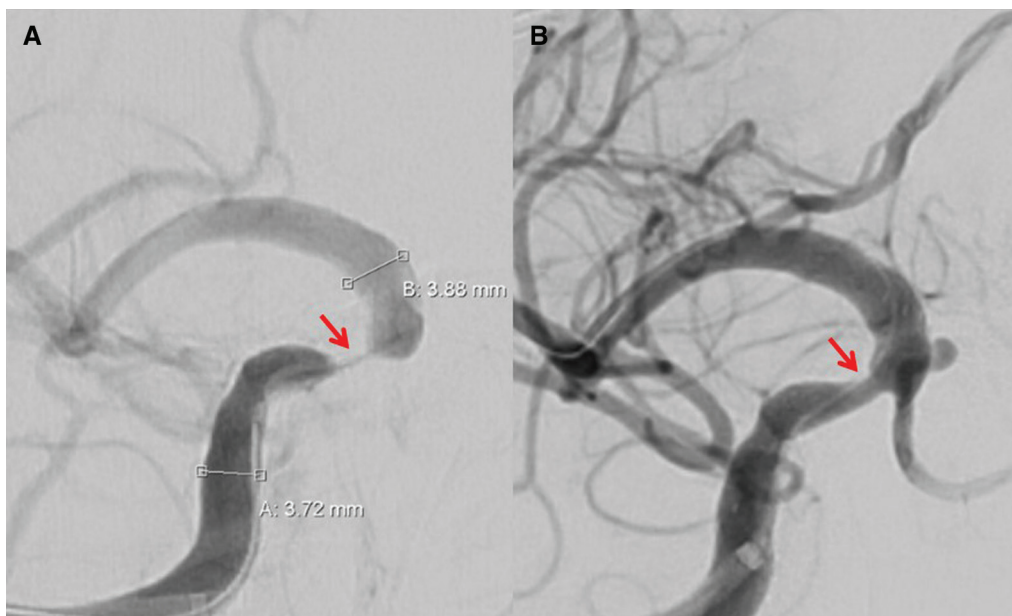


Fig. 3 Intraoperative findings of the intracranial stenting. (A) Right carotid angiogram immediately before the second surgery, showing progression of the stenosis to near-occlusion. (B) Right carotid angiogram after stenting, demonstrating successful treatment.

stenting.⁴⁻⁹ This report adds to the number of documented cases, with special emphasis on NIRS monitoring. Stenting for intracranial arteriosclerotic stenosis is associated with higher risks than pharmacotherapy alone according to a recent study.¹³ In Japan, the intracranial stent is indicated for the following conditions: (1) rescue of dissection and/or

acute occlusion occurring during percutaneous balloon angioplasty and (2) retreatment after angioplasty, with no other effective treatment. In the present case, restenosis despite aggressive pharmacotherapy after balloon angioplasty indicated that intracranial stenting was the cause of the cerebral HPS.

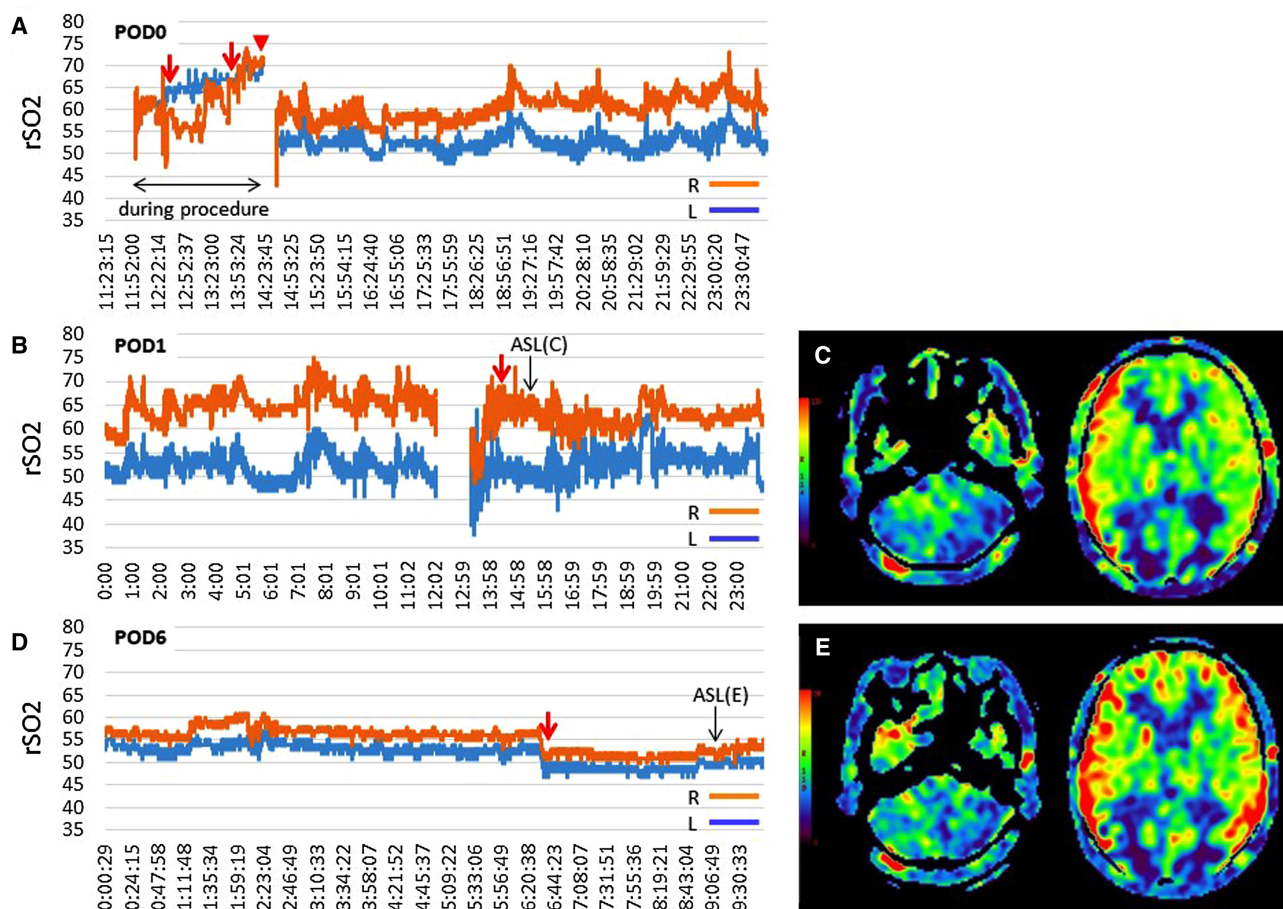


Fig. 4 Time course of the NIRS and ASL imaging after intracranial stenting. (A) NIRS graph plotted over the time course of intraoperative and ICU management showing right-sided increase from 60, before stenting, to 66, after successful stenting (arrows), while the values for the left hemisphere remained stable (around 66). The NIRS values from the right hemisphere showed the beginning of hyperperfusion when the NIRS cables were temporarily disconnected (arrow head). Plots after the arrow heads represent data in the ICU after stenting. Actual time is indicated in the transverse axis as [time:minute:second] (B) NIRS, continuing from (A) showing a further increase only on the affected side to 70/53 (arrow) 24 hours after stenting. (C) An ASL image obtained at the time indicated in (B) showing high signal intensity in the right middle cerebral artery territory compared to the contralateral side. (D) The laterality of NIRS improved to 53/50 (arrow) on the sixth day, postoperatively. (E) An ASL image obtained at the time indicated in (D) showing diminished right to left laterality, indicating improvement in hyperperfusion seen in (C). ASL: arterial spin labeling; ICU: intensive care unit; NIRS: near-infrared spectroscopy.

This HPS is thought to occur due to impaired cerebral autoregulation.¹⁴⁾ The incidence of HPS is about 1.9% after CEA and about 1.2% after CAS, according to systematic analyses of a large series of patients treated with CEA or CAS.³⁾ The risk factors for HPS include longstanding hypertension, increased age, high-grade ipsilateral stenosis, poor collateral flow, contralateral carotid occlusion, and postoperative hypertension.¹¹⁾ The present study reported hyperperfusion peaked on the sixth day after CEA and within 12 hours after CAS.³⁾ The time course of hyperperfusion has not been reported in cases with intracranial stenting, and this study may be the first report of the continuous postoperative monitoring of hyperperfusion.

In the present case, the stenotic lesion had progressed to near-occlusion and N-isopropyl-p-[¹²³I] iodoamphetamine SPECT revealed type 3 ischemia before the second surgery, suggesting that preoperative hemodynamic ischemia is a risk factor for post-stenting hyperperfusion in patients with

intracranial stenosis. NIRS is convenient for CBF monitoring as an alternative device to SPECT, which is less versatile. NIRS can be used easily and continuously to evaluate the approximate CBF condition from regional oxygen saturation (rSO₂), and there are several reports on the prediction of hyperperfusion after CEA or CAS during the procedure.¹⁵⁻¹⁷⁾ By performing continuous NIRS monitoring after surgery, as in the present case, not only the diagnosis of HPS but also the termination of the hyperperfusion can be accurately detected, which is effective for strict management. Particularly, in the elderly, adverse events due to long-term sedation are concerning; therefore, NIRS monitoring would be a good parameter to complete the treatment. However, NIRS monitors the rSO₂ of the ACA territory when the sensor is attached to the forehead, and it may underestimate the CBF if the A1 segment of the affected side is hypoplastic. The complementary use of NIRS, ASL, and SPECT may be efficacious for the monitoring, diagnosis, and precise timing of

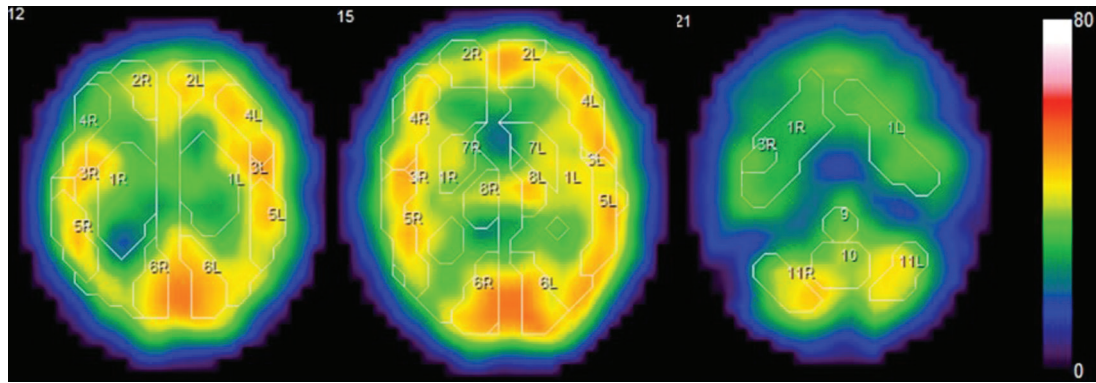


Fig. 5 Finding of the SPECT after intracranial stenting. No laterality was observed on N-isopropyl-p-[¹²⁵I] iodoamphetamine SPECT 20 days after the operation. SPECT: single-photon emission computed tomography.

treatment in patients with a high risk for postoperative hyperperfusion.

Conclusion

We document an unusual case of cerebral HPS after intracranial ICA stenting. Although the definite mechanism underlying this rare complication is unclear, CBF monitoring with NIRS, not only during the intracranial stenting procedure but also postoperatively, is useful for the strict management of HPS.

Conflicts of Interest Disclosure

The authors declare no conflict of interest.

References

- 1) Karapanayiotides T, Meuli R, Devuyt G, et al.: Postcarotid endarterectomy hyperperfusion or reperfusion syndrome. *Stroke* 36: 21–26, 2005
- 2) Meyers PM, Higashida RT, Phatouros CC, et al.: Cerebral hyperperfusion syndrome after percutaneous transluminal stenting of the cranio-cervical arteries. *Neurosurgery* 47: 335–343; discussion 343–345, 2000
- 3) Ogasawara K, Sakai N, Kuroiwa T, et al.: Intracranial hemorrhage associated with cerebral hyperperfusion syndrome following carotid endarterectomy and carotid artery stenting: retrospective review of 4494 patients. *J Neurosurg* 107: 1130–1136, 2007
- 4) Zhang R, Zhou G, Xu G, Liu X: Posterior circulation hyperperfusion syndrome after bilateral vertebral artery intracranial stenting. *Ann Vasc Surg* 23: 686.e1–5, 2009
- 5) Rezende MT, Spelle L, Mounayer C, Piotin M, Abud DG, Moret J: Hyperperfusion syndrome after stenting for intracranial vertebral stenosis. *Stroke* 37: e12–14, 2006
- 6) Xu S, Wu P, Shi H, Ji Z, Dai J: Hyperperfusion syndrome after stenting for intracranial artery stenosis. *Cell Biochem Biophys* 3: 1537–1542, 2015
- 7) Ghuman M, Tsang ACO, Klostranec JM, Krings T: Sentinel angiographic signs of cerebral hyperperfusion after angioplasty and stenting of intracranial atherosclerotic stenosis: a technical note. *AJNR Am J Neuroradiol* 40: 1523–1525, 2019
- 8) Meyers PM, Phatouros CC, Higashida RT: Hyperperfusion syndrome after intracranial angioplasty and stent placement. *Stroke* 37: 2210–2211, 2006
- 9) Medel R, Crowley RW, Dumont AS: Hyperperfusion syndrome following endovascular cerebral revascularization. *Neurosurg Focus* 26: E4, 2009
- 10) Samuels OB, Joseph GJ, Lynn MJ, Smith HA, Chimowitz MI: A standardized method for measuring intracranial arterial stenosis. *AJNR Am J Neuroradiol* 21: 643–646, 2000
- 11) Kuroda S, Kamiyama H, Abe H, Houkin K, Isobe M, Mitsumori K: Acetazolamide test in detecting reduced cerebral perfusion reserve and predicting long-term prognosis in patients with internal carotid artery occlusion. *Neurosurgery* 32: 912–918; discussion 918–919, 1993
- 12) Fujimura M, Niizuma K, Inoue T, et al.: Minocycline prevents focal neurological deterioration due to cerebral hyperperfusion after extracranial-intracranial bypass for moyamoya disease. *Neurosurgery* 74: 163–170; discussion 170, 2014
- 13) Derdeyn CP, Chimowitz MI, Lynn MJ, et al.: Aggressive medical treatment with or without stenting in high-risk patients with intracranial artery stenosis (SAMMPRIS): the final results of a randomised trial. *Lancet* 383: 333–341, 2014
- 14) Moulakakis KG, Mylonas SN, Sfyroeras GS, Andrikopoulos V: Hyperperfusion syndrome after carotid revascularization. *J Vasc Surg* 49: 1060–1068, 2009
- 15) Terakado T, Marushima A, Koyama Y, et al.: Effectiveness of near-infrared spectroscopy (NIRO-200NX, Pulse Mode) for risk management in carotid artery stenting. *World Neurosurg* 131: e425–e432, 2019
- 16) Pennekamp CW, Immink RV, den Ruijter HM, et al.: Near-infrared spectroscopy can predict the onset of cerebral hyperperfusion syndrome after carotid endarterectomy. *Cerebrovasc Dis* 34: 314–321, 2012
- 17) Matsumoto S, Nakahara I, Higashi T, et al.: Near-infrared spectroscopy in carotid artery stenting predicts cerebral hyperperfusion syndrome. *Neurology* 72: 1512–1518, 2009

Corresponding author:

Shuntaro Togashi, MD, PhD, Department of Neurosurgery, Akita University Graduate School of Medicine, 1-1-1 Hondo, Akita, Akita 010-8543, Japan.

✉togashi@med.akita-u.ac.jp