Intracerebral Hemorrhage Caused by Cerebral Hyperperfusion after Superficial Temporal Artery to Middle Cerebral Artery Bypass for Atherosclerotic Occlusive Cerebrovascular Disease

Fumihiro Matano,1 Yasuo Murai,1 Takayuki Mizunari,2 Koji Adachi,3 Shiro Kobayashi,2 and Akio Morita1

Few papers have reported detailed accounts of intracerebral hemorrhage caused by cerebral hyperperfusion after superficial temporal artery to middle cerebral artery bypass (STA-MCA) bypass for atherosclerotic occlusive cerebrovascular disease. We report a case of vasogenic edema and subsequent intracerebral hemorrhage caused by the cerebral hyperperfusion syndrome (CHS) after STA-MCA bypass for atherosclerotic occlusive cerebrovascular disease without intense postoperative blood pressure control. A 63-year-old man with repeating left hemiparesis underwent magnetic resonance angiography (MRA), which revealed right internal carotid artery (ICA) occlusion. We performed a double bypass superficial temporal artery (STA)–middle cerebral artery (MCA) bypass surgery for the M2 and M3 branches. While the patient’s postoperative course was relatively uneventful, he suffered generalized convulsions, and computed tomography revealed a low area in the right frontal lobe on Day 4 after surgery. We considered this lesion to be pure vasogenic edema caused by cerebral hyperperfusion after revascularization. Intravenous drip infusion of a free radical scavenger (edaranavone) and efforts to reduce systolic blood pressure to <120 mmHg were continued. The patient experienced severe left hemiparesis and disturbance of consciousness on Day 8 after surgery, due to intracerebral hemorrhage in the right frontal lobe at the site of the earlier vasogenic edema. Brain edema associated with cerebral hyperperfusion after STA-MCA bypass for atherosclerotic occlusive cerebrovascular disease should be recognized as a risk factor for intracerebral hemorrhage. The development of brain edema associated with CHS after STA-MCA bypass for atherosclerotic occlusive cerebrovascular disease requires not only intensive control of blood pressure, but also consideration of sedation therapy with propofol.

Keywords: atherosclerotic; cerebral hyperperfusion; intracerebral hemorrhage; extracranial–intracranial bypass

Received: February 15, 2016; Accepted: July 10, 2016
Fig. 1  (A) Pre-operative magnetic resonance angiography (MRA) revealed right internal carotid artery (ICA) occlusion. (B) Post-operative MRA revealed good bypass patency.

intensity when T₂-weighted (Fig. 5A) and slightly higher intensity when diffusion-weighted (Fig. 5B); and perfusion CT revealed hyperperfusion (Fig. 3B). We considered this lesion to be pure vasogenic edema caused by cerebral hyperperfusion after revascularization. Intravenous drip infusion of a free radical scavenger (edaravone) was continued, and systolic blood pressure was kept below 120 mmHg. The patient continued to experience severe left hemiparesis and disturbance of consciousness on Day 8 after surgery, due to intracerebral hemorrhage in the right frontal lobe at the site of the earlier vasogenic edema (Fig. 4C). We stopped anti-platelet therapy and continued to reduce systolic blood pressure. Follow up CT and MRI revealed no expanding hematoma or edema. The patient was transferred to another hospital for rehabilitation.

Fig. 2  Single-photon emission computed tomography (SPECT) scan revealed a perfusion defect in the right frontal (hypoperfusion) and decreased cerebrovascular reactivity in the right hemisphere on the pre-operative with acetazolamide challenge test.
Discussion

We report a case of vasogenic edema and subsequent intracerebral hemorrhage caused by CHS after STA-MCA bypass for atherosclerotic occlusive cerebrovascular disease without intense postoperative blood pressure control. Brain edema associated with CHS after STA-MCA bypass for atherosclerotic occlusive cerebrovascular disease should be recognized as a risk factor of ICH.

Vascular reconstruction surgery for patients with intracranial hypoperfusion can cause a rapid increase in cerebral blood flow, thereby resulting in CHS, which is characterized by unilateral headache, face and eye pain, seizures, and focal symptoms related to cerebral edema or intracranial hemorrhage.8,9)

Several methods of vascular reconstruction have been developed to treat ischemic cerebrovascular diseases, and the frequency of hyperperfusion varies with each, CHS

Fig. 3  (A) Pre-operative perfusion CT revealed at resting state revealed a perfusion defect (hypoperfusion) in the right frontal area of the right hemisphere. (B) Perfusion CT revealed hyperperfusion on Day 5 after surgery.

Fig. 4  (A) CT revealed no abnormalities on Day 1 after surgery. (B) CT revealed a low area in a right frontal on Day 4 after surgery. (C) CT revealed the right frontal intracerebral hemorrhage at the corresponding lesion to the prior vasogenic edema on Day 8 after surgery.
and ICH caused by CHS occasionally occur in patients after carotid endarterectomy (CEA), carotid artery stenting (CAS), and STA-MCA bypass for moyamoya disease, but CHS after STA-MCA bypass for atherosclerotic occlusive cerebrovascular disease is relatively rare and typically manifests as mild focal neurological deficit. Further, no detailed reports have been published on intracerebral hemorrhage caused by cerebral hyperperfusion after revascularization for atherosclerotic occlusive cerebrovascular disease.

Patients with poorer cerebrovascular reactivity and weakness of the blood-brain barrier due to chronic ischemia are known to have a potentially higher risk of hyperperfusion syndrome (HPS). The increase of vascular endothelial growth factor and poorer network formation between the pial arteries in moyamoya disease contributes to the vulnerability to CHS in moyamoya patients compared to those with atherosclerotic disease. In the present case, MRA revealed right ICA occlusion, and a SPECT scan showed an extensive area of hypoperfusion and decreased cerebrovascular reactivity in the right hemisphere. Given the above and the fact that we performed a double bypass for the M2 and M3 branches and not the M4 of cortical branch, the area of poorer cerebrovascular reactivity would receive a greater blood supply than is usual after bypass.

It may have caused the HPS and ICH observed in the present case. In fact, a previous report showed that STA-M4 bypass seldom resulted in post-operative HPS. Therefore, double anastomoses and M2 or M3 bypass may induce excessive flow and single bypass or M4 bypass should be appropriate.

Edema associated with HPS in the early stage is considered to be vasogenic edema. Typical vasogenic edema appears as an area of low signal intensity on diffusion-weighted MRI. This was the case in our patient, where hyperperfusion occurred, the right frontal lobe at the site of anastomosis appeared as a low intensity lesion on CT, as a high-intensity area on T2-weighted MRI, and as a low-intensity area on diffusion-weighted MRI, findings which were typical of vasogenic edema.

Fujimura et al. reported that a patient with moyamoya disease who underwent STA-MCA developed vasogenic edema due to HPS. Despite the patient’s blood pressure being strictly controlled to less than 120 mmHg, the edema progressed to intracerebral hemorrhage. These authors concluded that an early increase in cerebral blood flow associated with vasogenic edema formation at the site of anastomosis may be a warning sign for subsequent hemorrhagic complications. We observed a similar clinical course with the present case and drew a similar conclusion regarding STA-MCA bypass for atherosclerotic occlusive cerebrovascular disease. In our case, we controlled blood pressure to less than 140 mmHg from immediately after operation to the development of vasogenic edema, but this resulted in the development of critical vasogenic edema. Intensive anti-hypertensive treatment (systolic blood pressure < 130 mmHg) immediately after STA-MCA bypass surgery prevents HPS in moyamoya disease, suggesting the need for strict postoperative control of blood pressure in atherosclerotic occlusive cerebrovascular disease.

Seizure due to HPS has been reported as a contraindication for anticoagulation therapy due to the increased risk of hemorrhage in such patients. Continuous sedation is therefore recommended for the first 48 h post-surgery to prevent hyperperfusion.

Fig. 5  (A) Magnetic resonance imaging (MRI) revealed the high intensity on T2-weight on the day after Day 5 after surgery. (B) Magnetic resonance imaging (MRI) revealed slightly high on diffusion on the day after Day 5 after surgery.
Although these studies targeted CEA, we should consider the use of propofol sedation therapy, which reduces lactate production and cerebral metabolic rate when brain edema associated with CSH appears. 30

Conclusion
We report a case of vasogenic edema following intracerebral hemorrhage caused by CSH after STA-MCA bypass for atherosclerotic occlusive cerebrovascular disease without intense postoperative blood pressure control. The use of propofol sedation therapy to prevent ICH due to CSH should be considered in patients developing vasogenic edema.

Conflicts of Interest Disclosure
The authors have no conflicts of interest. All authors who are members of the Japan Neurosurgical Society (JNS) have registered on line Self-reported COI Disclosure Statement Forms through the website for JNS members.

References
1) Fluri F, Engelter S, Lyer P: Extracranial-intracranial arterial bypass surgery for occlusive carotid artery disease. Cochrane Database Syst Rev CD003953, 2010
2) Holohan TV: Extracranial-intracranial bypass to reduce the risk of ischemic stroke. CMAJ 144: 1457–1465, 1991
3) Murai Y, Mizunari T, Takagi R, Amano Y, Mizumura S, Komaba Y, Okubo S, Kobayashi S, Teramoto A: Analysis of ischemic cerebral lesions using 3.0-T diffusion-weighted imaging and magnetic resonance angiography after revascularization surgery for ischemic disease. Clin Neurol Neurosurg 115: 1063–1070, 2013
4) Fujimura M, Shimizu H, Inoue T, Mugikura S, Saito A, Tominağa T: Significance of focioc cerebral hyperperfusion as a cause of transient neurologic deterioration after extracranial-intracranial bypass for moyamoya disease: comparative study with non-moyamoya patients using N-isopropyl-p-[123]Ijodoamphetamine single-photon emission computed tomography. Neurosurgery 68: 957–964, 2011
5) Hosoda K, Kawaguchi T, Ishii K, Minoshima S, Shibata Y, Iwakura M, Ishiguro S, Kohmura E: Prediction of hyperperfusion after carotid endarterectomy by brain SPECT analysis with semiquantitative statistical mapping method. Stroke 34: 1187–1193, 2003
6) Narisawa A, Fujimura M, Shimizu H and Tominağa T: [Seizure following superficial temporal-middle cerebral artery anastomosis in patients with moyamoya disease: possible contribution of postoperative cerebral hyperperfusion], No Shinketsu Geka 35: 467–474, 2007
7) Fujimura M, Mugikura S, Kaneta T, Shimizu H, Tominağa T: Incidence and risk factors for symptomatic cerebral hyperperfusion after superficial temporal artery-middle cerebral artery anastomosis in patients with moyamoya disease. Surg Neurol 71: 442–447, 2009
8) Sundt TM, Sandok BA, Whisnant JP: Carotid endarterectomy, complications and preoperative assessment of risk. Mayo Clin Proc 50: 301–306, 1975
9) Piepers DG, Morgan MK, Sundt TM, Yanagihara T, Musmann LM: Intracerebral hemorrhage after carotid endarterectomy. J Neurosurg 68: 532–536, 1988
10) Gröschel K, Riecker A, Schulz JB, Ernemann U, Kastrup A: Systematic review of early recurrent stenosis after carotid angioplasty and stenting. Stroke 36: 367–373, 2005
11) Halliday A, Mansfield A, Marro J, Peto C, Peto R, Potter J, Thomas D: MRC Asymptomatic Carotid Surgery Trial (ACST) Collaborative Group: Prevention of disabling and fatal strokes by successful carotid endarterectomy in patients without recent neurological symptoms: randomised controlled trial. Lancet 363: 1491–1502, 2004
12) Katano H, Mase M, Sakurai K, Miyachi S, Yamada K: Revascularization of collateral pathways as escape routes from hyperemia/hyperperfusion following surgical treatment for carotid stenosis. Acta Neurochir (Wien) 154: 2139–2148, 2012
13) Yoshimura S, Kitajima H, Enomoto Y, Yamada K, Iwama T: Staged angioplasty for carotid artery stenosis to prevent postoperative hyperperfusion. Neurosurgery 64: ons122–ons128; discussion ons128–ons129, 2009
14) Sundt TM, Sharbrough FW, Piepgras DG, Kearns TP, Messick JM, O’Fallon WM: Correlation of cerebral blood flow and electroencephalographic changes during carotid endarterectomy: with results of surgery and hemodynamics of cerebral ischemia. Mayo Clin Proc 56: 553–543, 1981
15) Ogasawara K, Sakai N, Kurosawa T, Hosoda K, Iiura H, Toyoda K, Sakai C, Nagata I, Ogawa A: Japanese Society for Treatment at Neck in Cerebrovascular Disease Study Group: 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
16) Hafner DH, Smith RB, King OW, Perdue GD, Stewart MT, Rosenthal D, Jordan WD: Massive intracerebral hemorrhage following carotid endarterectomy. Arch Surg 122: 305–307, 1987
17) Solomon RA, Loftus CM, Quest DO, Correll JW: Incidence and etiology of intracerebral hemorrhage following carotid endarterectomy. J Neurosurg 64: 29–34, 1986
18) Abou-Chbel A, Yadav JS, Regnellini JP, Bajzer C, Bhatt D, Krieger DW: Intracranial hemorrhage and hyperperfusion syndrome following carotid artery stenting: risk factors, prevention, and treatment. J Am Coll Cardiol 43: 1596–1601, 2004
19) Wu TY, Anderson NE, Barber PA: Neurological complications of carotid revascularisation. J Neurol Neurosurg Psychiatry 83: 543–550, 2012
20) van Mook WN, Rennenberg RJ, Schurink GW, van Oostenbrugge RJ, Mess WH, Hofman PA, de Leeuw PW: Cerebral hyperperfusion syndrome. Lancet Neurol 4: 777–888, 2005
21) Ohnishi K, Kohno K, Watanabe H, Iwata S, Ohsnishi T: Postoperative temporary neurological deficits in adults with moyamoya disease. Surg Neurol 69: 281–286; discussion 286–287, 2008
22) Heros RC, Scott RM, Kistler JP, Ackerman RH, Conner ES: Temporal neurological deterioration after extracranial-intracranial bypass. Neurosurgery 15: 178–185, 1984
23) Higashi S, Matsuoka H, Fuji H, Ito H, Yamashita J: Luxury perfusion syndrome confirmed by sequential studies of regional cerebral blood flow and volume after extracranial to intracranial bypass surgery: case report. Neurosurgery 25: 85–89, 1989
24) Kuroda S, Kamiyama H, Abe H, Asaoka K, Mtsumori K: Temporary neurological deterioration caused by hyperperfusion after extracranial-intracranial bypass—case report and study of cerebral hemodynamics. Neurol Med Chir (Tokyo) 34: 15–19, 1994
25) Uno M, Nakajima N, Nishi K, Shinno K, Nagahiro S: Hyperperfusion syndrome after extracranial-intracranial bypass in a patient with moyamoya disease—case report. Neurol Med Chir (Tokyo) 38: 420–424, 1998
26) Ogasawara K, Yukawa H, Kobayashi M, Mikami C, Konno H, Terasaki K, Inoue T, Ogawa A: Prediction and monitoring of cerebral hyperperfusion after carotid endarterectomy by using single-photon emission computed tomography scanning. J Neurosurg 99: 504–510, 2003
27) Yamaguchi K, Kawamata T, Kawashima A, Hori T, Okada Y: Incidence and predictive factors of cerebral hyperperfusion after extracranial-intracranial bypass for occlusive cerebrovascular diseases. Neurosurgery 67: 1548–1554; discussion 1554, 2010
28) Kim JE, Oh CW, Kwon OK, Park SQ, Kim SE, Kim YK: Transient hyperperfusion after superficial temporal artery/middle cerebral artery bypass surgery as a possible cause of postoperative transient neurologic deterioration. Cerebrovasc Dis 25: 580–586, 2008
29) Fujimura M, Watanabe M, Narisawa A, Shimizu H, Tominağa T: Increased expression of serum Matrix Metalloproteinase-9 in patients with moyamoya disease. Surg Neurol 72: 476–480; discussion 480, 2009
30) Ay H, Buonanno FS, Schaefer PW, Le DA, Wang B, Gonzalez RG, Koroshetz WJ: Posterior leukoencephalopathy without severe hypotension: utility of diffusion-weighted MRI. Neurology 51: 1369–1376, 1998
31) Casey SO, Truwit CL: Pontine reversible edema: a newly recognized imaging variant of hypertensive encephalopathy? AJNR Am J Neuroradiol 21: 243–245, 2000
32) Covernubrias DJ, Luechter PH, Campeau NG: Posterior reversible encephalopathy syndrome: prognostic utility of quantitative diffusion-weighted MR images. AJNR Am J Neuroradiol 23: 1038–1048, 2002
33) Kokuzawa J, Kaku Y, Watarai T, Tanaka T, Hatasu N, Ando T: Pure vasogenic edema caused by cerebral hyperperfusion after superficial
temporal artery to middle cerebral artery anastomosis—case report.

34) Fujimura M, Shimizu H, Mugikura S, Tominaga T: Delayed intracerebral hemorrhage after superficial temporal artery-middle cerebral artery anastomosis in a patient with moyamoya disease: possible involvement of cerebral hyperperfusion and increased vascular permeability. Surg Neurol 71: 223–227; discussion 227, 2009

35) Fujimura M, Inoue T, Shimizu H, Saito A, Mugikura S, Tominaga T: Efficacy of prophylactic blood pressure lowering according to a standardized postoperative management protocol to prevent symptomatic cerebral hyperperfusion after direct revascularization surgery for moyamoya disease. Cerebrovasc Dis 33: 436–445, 2012

36) Kawamata T, Okada Y, Kawashima A, Yoneyama T, Yamaguchi K, Ono Y, Hori T: Postcarotid endarterectomy cerebral hyperperfusion can be prevented by minimizing intraoperative cerebral ischemia and strict postoperative blood pressure control under continuous sedation. Neurosurgery 64: 447–453, 2009

37) Kalimeris K, Kouni S, Kostopanagiotou G, Nomikos T, Fragopoulou E, Kakisis J, Vasdekis S, Matsota P, Pandazi A: Cognitive function and oxidative stress after carotid endarterectomy: comparison of propofol to sevoflurane anesthesia. J Cardiothorac Vasc Anesth 27: 1246–1252, 2013

38) Lao N, Nie H, Xu LX, Xiong LZ, Zhang H, Fan YY, Wang BR: Efficacy of intracarotid propofol infusion and impact of cerebral blood flow alteration. Br J Anaesth 102: 234–239, 2009

Corresponding author:
Fumihiro Matano, Department of Neurosurgery, Chiba Hokusoh Hospital, 1715 Kamagari, Inzai, Chiba, 270-1694, Japan.
✉️ s00-078@nms.ac.jp