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Case Report

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 Morita[1]

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 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 (edaravone) 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

[1]Department of Neurological Surgery, Nippon Medical School, Tokyo, Japan
[2]Department of Neurosurgery, Chiba Hokusoh Hospital, Chiba, Japan
[3]Department of Neurosurgery, Nippon Medical School Musashi Kosugi Hospital, Kanagawa, Japan

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Introduction

Superficial temporal artery to middle cerebral artery (STA-MCA) bypass surgery for cerebrovascular ischemia, such as that caused by moyamoya disease and atherosclerotic occlusive disease was effective in improving blood flow to the brain to prevent stroke.[1–3] However, vascular reconstructive surgery may cause a rapid increase in cerebral blood flow, resulting in cerebral hyperperfusion syndrome (CHS).

Previous studies have reported that CHS is more frequent after STA-MCA bypass for moyamoya disease than after that for atherosclerotic disease,[4] and intracerebral hemorrhage (ICH) caused by CHS after STA-MCA bypass for moyamoya disease has also been reported.[5–7] In contrast, few studies have reported ICH caused by CHS after STA-MCA bypass for atherosclerotic occlusive cerebrovascular disease. Here, we describe a case of ICH caused by CHS after STA-MCA bypass for atherosclerotic occlusive cerebrovascular disease and review the related literature on this topic.

Case Report

A 63-year-old man with a history of hypertension presented at our hospital with repeated left hemiparesis. While a neurological examination at our facility revealed no abnormalities, magnetic resonance angiography (MRA) revealed right internal carotid artery occlusion (Fig. 1A). A single-photon emission computed tomography (SPECT) scan, with and without acetazolamide challenge, revealed a perfusion defect in the right frontal lobe (hypoperfusion) and decreased cerebrovascular reactivity in the right hemisphere (Fig. 2). Perfusion computed tomography (CT) revealed hypoperfusion in the right frontal lobe (Fig. 3A).

We performed STA-MCA bypass surgery. Briefly, we dissected the STA during a craniotomy, and after incising through the dura mater and opening the Sylvian fissure, we selected an appropriate recipient segment, performing a double bypass for the M2 and M3 branches. Intra-operative trapping time was 27 min and 22 min. The patient's postoperative course was relatively uneventful, and CT revealed no abnormalities (Fig. 4A). However, the patient suffered generalized convulsions, and CT revealed a low-density area in the right frontal lobe on Day 4 after surgery (Fig. 4B). On Day 5 after surgery, MRA revealed good bypass patency (Fig. 1B); magnetic resonance imaging (MRI) revealed high

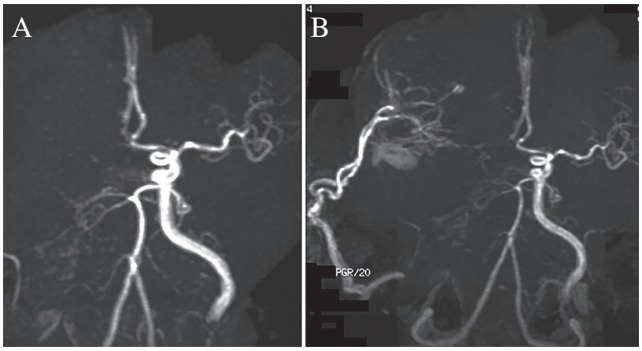

Fig. 1 (A) Pre-operative magnetic resonance angiography (MRA) revealed right internal carotid artery (ICA) occlusion. (B) Post-operative MRA revealed good bypass patency.

intensity when T_2-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.

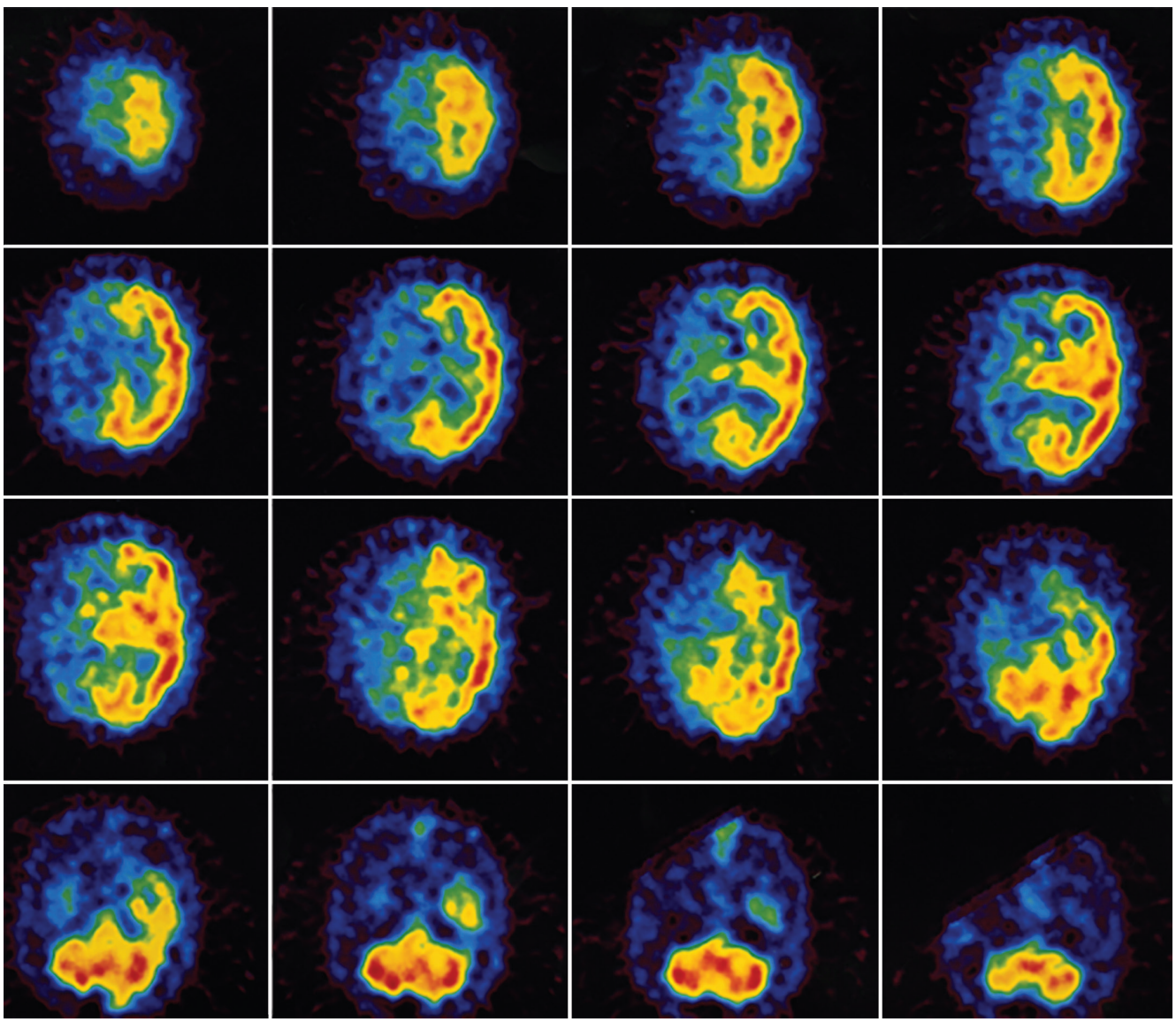

Fig. 2 Single-photon emission computed tomography (SPECT) scan revealed a perfusion defect in the right frontal (hypoperfusion) and decreased cerebrovascular reactivity in the right hemisphere on the pre-operative with acetazolamide challenge test.

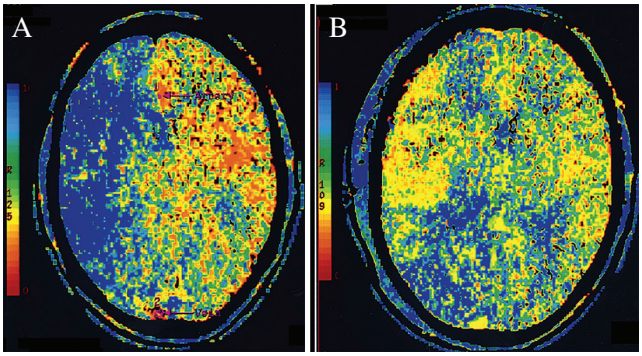

Fig. 3 (A) Pre-operative perfusion CT revealed at resting state revealed a perfusion defect (hypoperfusion) in the right frontal area of the right hemisphere. (B) Perfusion CT revealed hyperperfusion on Day 5 after surgery.

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,[10,11] and the frequency of hyperperfusion varies with each,[9,12–18] CHS

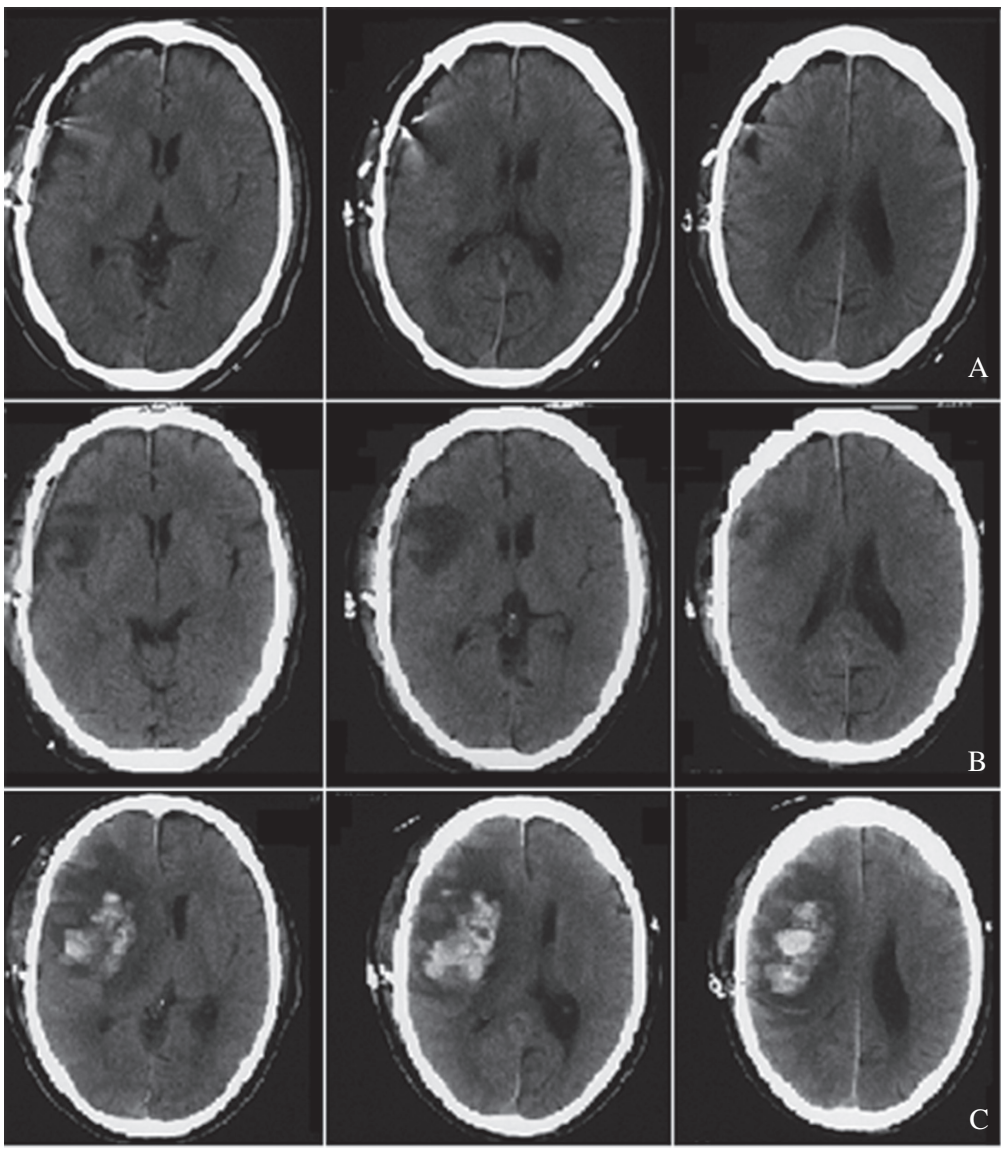

Fig. 4 (A) CT revealed no abnormalities on Day 1 after surgery. (B) CT revealed a low area in a right frontal on Day 4 after surgery. (C) CT revealed the right frontal intracerebral hemorrhage at the corresponding lesion to the prior vasogenic edema on Day 8 after surgery.

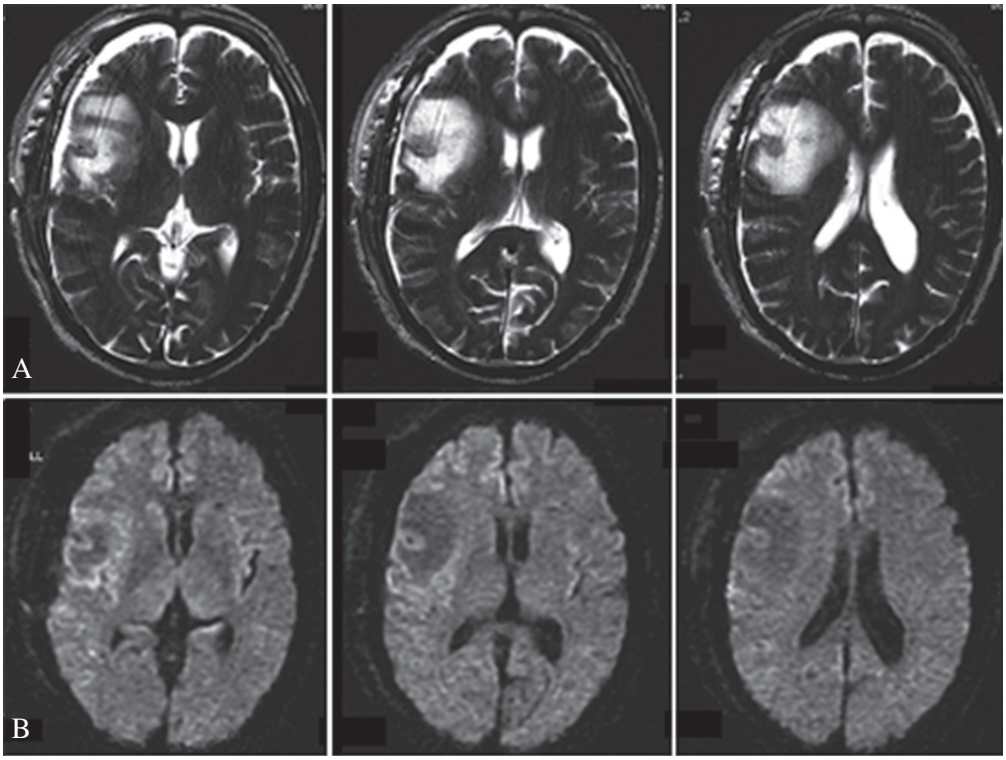

Fig. 5 (A) Magnetic resonance imaging (MRI) revealed the high intensity on T_2-weight on the day after Day 5 after surgery. (B) Magnetic resonance imaging (MRI) revealed slightly high on diffusion on the day after Day 5 after surgery.

and ICH caused by CHS occasionally occur in patients after carotid endarterectomy (CEA), carotid artery stenting (CAS), and STA-MCA bypass for moyamoya disease,[7,9,14,17,19–21] but CHS after STA-MCA bypass for atherosclerotic occlusive cerebrovascular disease is relatively rare[22–25] 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)[20,26,27] 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.[28,29] 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[4] 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.[30–32]

Typical vasogenic edema appears as an area of low signal intensity on diffusion-weighted MRI.[33] In our case, when 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 T_2-weighted MRI, and as a low-intensity area on diffusion-weighted MRI, findings which were typical of vasogenic edema.

Fujimura et al.[34] 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,[35] 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.[9] Continuous sedation is therefore recommended for the first 48 h post-surgery to prevent hyperperfusion.[36]

Although these studies targeted CEA, we should consider the use of propofol sedation therapy, which reduces lactate production and cerebral metabolic rate[37] when brain edema associated with CHS appears.[36,38]

Conclusion

We report a case of vasogenic edema following intracerebral hemorrhage caused by CHS 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 CHS 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.

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