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**Pure Vasogenic Edema Caused by Cerebral
Hyperperfusion After Superficial Temporal
Artery to Middle Cerebral Artery Anastomosis
—Case Report—**

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Abstract

A 63-year-old patient presented with cerebral hyperperfusion manifesting as transient aphasia and general tonic convulsions 3 and 4 days after superficial temporal artery to middle cerebral artery (STA-MCA) anastomosis. Diffusion-weighted magnetic resonance (MR) imaging revealed a focal low-intensity area at the site of anastomosis in the left temporal lobe, with high apparent diffusion coefficient, together with focal intense increase in cerebral blood flow in the same region. This lesion was considered to be pure vasogenic edema caused by cerebral hyperperfusion. Additional treatment with intravenous drip infusion of free radical scavenger and reduction in blood pressure with nicardipine improved the patient's symptoms and brain edema. The brain edema gradually decreased on MR imaging and completely disappeared at 3 months after bypass surgery. Cerebral hyperperfusion is often encountered after recanalization of occlusive arteries, removal of arteriovenous malformations, and carotid endarterectomy, but may also occur after STA-MCA anastomosis.

Key words: hyperperfusion, superficial temporal artery-middle cerebral artery anastomosis, vasogenic edema, diffusion-weighted magnetic resonance imaging, free radical scavenger

Introduction

Cerebrovascular reconstructive surgery for patients with atherosclerotic cerebral occlusive disease can cause a rapid increase in cerebral blood flow (CBF) in the chronic ischemic brain, resulting in cerebral hyperperfusion with brain edema and hemorrhage. Cerebral hyperperfusion syndrome sometimes occurs in patients after carotid endarterectomy,^{1,3,9-11,20} but is rare after superficial temporal artery to middle cerebral artery (STA-MCA) anastomosis.^{8,18,19} Based on theoretical considerations, the edema associated with cerebral hyperperfusion in the early stage is considered to be vasogenic edema.^{2,4,6,7} Typical vasogenic edema appears as low signal intensity on diffusion-weighted magnetic resonance (MR) imaging with high apparent diffusion coefficient (ADC) values.¹⁶ However, vasogenic and cytotoxic edema frequently occur together in patients with cerebral hyperperfusion.^{5-7,11}

We describe a case of pure vasogenic edema caused by cerebral hyperperfusion as demonstrated by MR imaging after STA-MCA anastomosis.

Case Report

A 63-year-old right-handed man had a medical history of hypertension and hyperlipidemia. Asymptomatic left MCA occlusion had been detected 10 years previously at another hospital. He visited our institution complaining of several recent episodes of aphasia. Diffusion-weighted MR imaging demonstrated no ischemic lesion, and MR angiography demonstrated left MCA occlusion (Fig. 1A, B). Technetium-99m ethyl cysteinate dimer single photon emission computed tomography (^{99m}Tc-ECD SPECT) showed that resting regional CBF of the left MCA territory was 10% lower compared to the right MCA territory (Fig. 1C). Furthermore, ^{99m}Tc-ECD SPECT with acetazolamide challenge showed severely impaired cerebrovascular reserve capacities in the territory of the left MCA ($\{[\text{CBF post acetazolamide} - \text{rest CBF}] / \text{rest CBF}\} \times 100\% = 12\%$) (Fig. 1D, E). He was considered a candidate for left STA-MCA anastomosis.

After exploration of the parietal branch of the left STA with a 5 cm linear skin incision, a 3 × 3 cm craniotomy was performed. The M₄ segment of the left MCA in the left temporal lobe was used as the recipient artery. End-to-side anastomosis was performed between the stump of the STA and the M₄ segment of the MCA. The patient developed no neurological deficits and had normal blood pressure for 2 days after surgery. He developed moderate headache and sensory-dominant dysphasia on the 3rd day after surgery with elevated systolic blood pressure up to 180 mmHg. He suffered generalized convulsions on the 4th day after surgery. Diffusion-weighted MR imaging revealed a focal low signal intensity area in the left temporal lobe, with increased ADC values in the same region (Fig. 2A, B). ^{99m}Tc-ECD SPECT demonstrated a focal intense increase in CBF at the site of the anastomosis (Fig. 2D). 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 reduc-

tion of systolic blood pressure to less than 120 mmHg with nicardipine were continued for the following 11 days. His symptoms improved within 24 hours. The subsequent clinical course was uneventful, and the lesion gradually decreased in size on MR imaging and finally disappeared by 3 months after surgery (Fig. 3).

Discussion

Cerebral hyperperfusion is classically defined as increased CBF compared with normal CBF in the presence of oversupply of blood exceeding tissue requirements. Cerebral hyperperfusion syndrome is characterized by unilateral headache, facial and ocular pain, seizures, and focal symptoms that occur secondary to cerebral edema or intracerebral hemorrhage.^{14,17,18} The cerebral edema usually presents as both vasogenic and cytotoxic edema on clinical neuroimaging.^{7,11} Clinically, cerebral hyperperfusion is often encountered after recanalization of occlusive arteries, removal of arteriovenous malformations,⁷ and carotid endarterectomy.^{1,3,9-11,20} However, cerebral hyperperfusion after STA-MCA anastomosis is rare.^{8,18,19}

SPECT CBF study is recommended for the correct diagnosis of postoperative cerebral hyperperfusion, since treatment of this condition differs from that for ischemia. MR imaging, particularly diffusion-weighted imaging, may be also important for the detection of cerebral injury due to cerebral hyperperfusion. Lesions with low diffusion-weighted signal intensity and high ADC values are considered to be pure vasogenic edema and reversible.¹² Vasogenic edema originates in hyperperfusion, resulting in failure of autoregulation and disruption of the blood-brain barrier, finally causing vasogenic edema,⁴ though the associated brain cell injury is not irreversible. Diffusion-weighted imaging of patients with cerebral hyperperfusion has suggested that vasogenic and cytotoxic edema are frequently present simultaneously.^{5-7,11} Our patient presented with low diffusion-weighted signal intensity at the site of the anastomosis and high ADC values in the same region, with focal intense increase in CBF at the site of the anastomosis. This lesion disappeared on follow-up MR imaging, together with normalization of regional CBF on follow-up SPECT. Therefore, we consider this lesion to be pure vasogenic edema caused by cerebral hyperperfusion after STA-MCA anastomosis. Patients presenting with pure vasogenic edema caused by cerebral hyperperfusion after revascularization are very rare, and this condition has never been associated with STA-MCA anastomosis.

Vasogenic edema can occur in the early stage of cerebral hyperperfusion. Rapid and profound hyperperfusion, such as that following carotid endarterectomy, causes massive vasogenic edema, and increased tissue pressure eventually impairs the microcirculation, leading to cytotoxic edema and irreversible cell injury. In contrast, STA-MCA anastomosis usually provides low-flow revascularization due to the anatomy of the recipient arteries, and cerebral hyperperfusion following STA-MCA anastomosis deteriorates more slowly, allowing detection of vasogenic edema. Therefore, treatment is required during

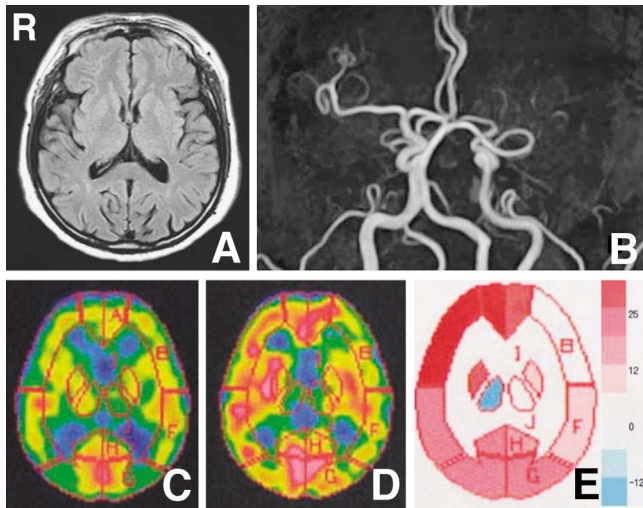


Fig. 1 A: Preoperative axial fluid-attenuated inversion recovery magnetic resonance (MR) image demonstrating absence of ischemic lesions. B: MR angiogram demonstrating left middle cerebral artery (MCA) occlusion. C: Technetium-99m ethyl cysteinate dimer single photon emission computed tomography (^{99m}Tc-ECD SPECT) scan showing a slight decrease in resting cerebral blood flow value in the area of the left MCA territory. D, E: ^{99m}Tc-ECD SPECT scans with acetazolamide challenge showing that markedly impaired cerebrovascular reserve capacities in the area of the left MCA on the percentage increase map.

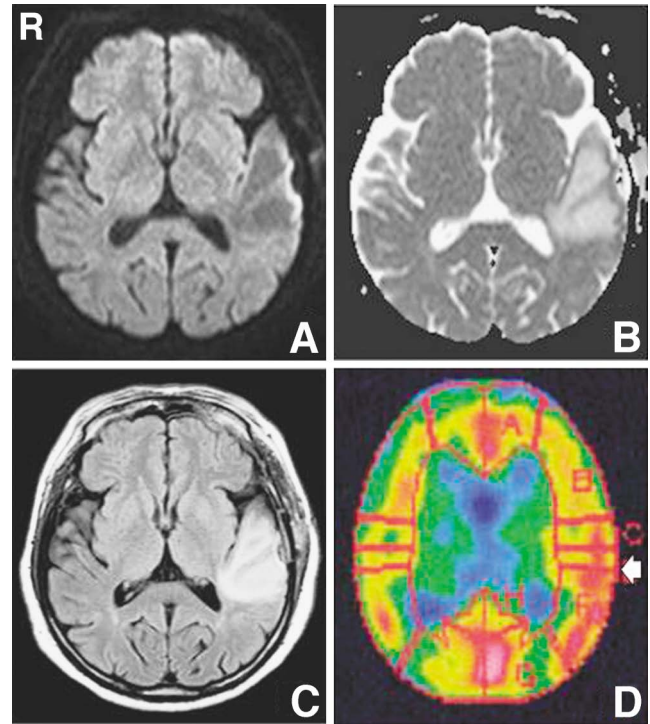


Fig. 2 A-C: Diffusion-weighted magnetic resonance (MR) images revealing low signal intensity in the left temporal region (A) with increased apparent diffusion coefficient values (B), and fluid-attenuated inversion recovery MR image demonstrating high signal intensity in the same region (C) 4 days after surgery. D: Technetium-99m ethyl cysteinate dimer single photon emission computed tomography scan revealing focal increase in cerebral blood flow at the site of the anastomosis (arrow).

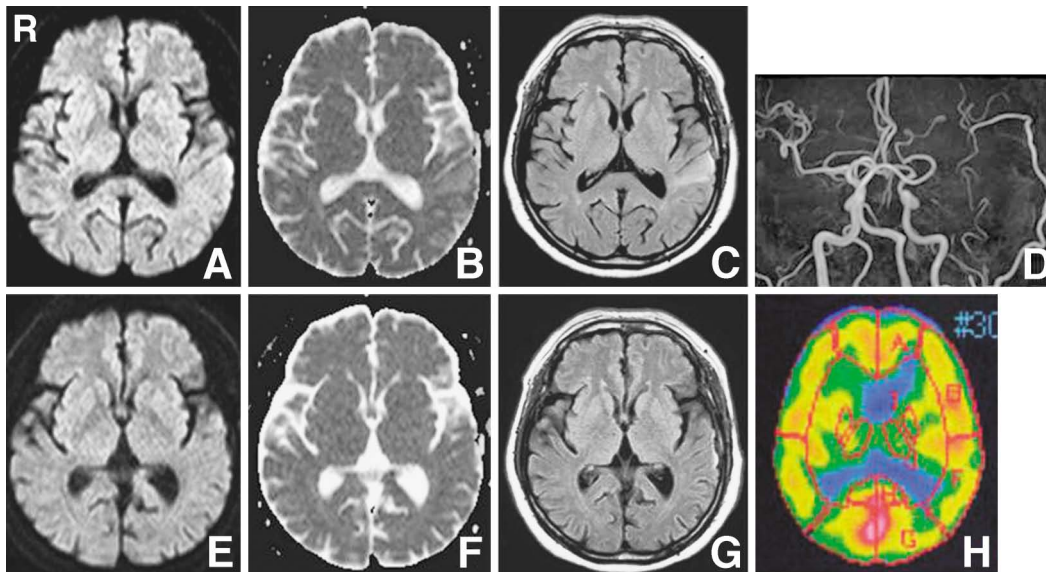


Fig. 3 A-C: Diffusion-weighted magnetic resonance (MR) image (A), apparent diffusion coefficient (ADC) map (B), and fluid-attenuated inversion recovery (FLAIR) MR image (C) showing the vasogenic edema had gradually decreased at 14 days. D: MR angiogram demonstrating patency of the superficial temporal artery-middle cerebral artery anastomosis. E-G: Diffusion-weighted MR image (E), ADC map (F), and FLAIR MR image (G) showing no vasogenic edema or brain damage at 3 months. H: Technetium-99m ethyl cysteinate dimer single photon emission computed tomography scan obtained 3 months after surgery demonstrating normalized cerebral blood flow of the lesion.

the stage of vasogenic edema to prevent progression to cytotoxic edema. Such treatments include intensive blood pressure control, and barbiturate or propofol coma therapy. Recently, antioxidant agents have been reported to prevent cerebral hyperperfusion syndrome after carotid endarterectomy.¹³ We treated our patient with a free radical scavenger to ameliorate the unfavorable effects of hyperperfusion on the brain.

Cerebral hyperperfusion syndrome after STA-MCA anastomosis is rare, but routine CBF study is useful for the correct diagnosis of postoperative hyperperfusion, and diffusion-weighted imaging may be useful for determination of brain damage in affected patients. Additional treatment must be performed while the patient's condition is still reversible.

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