



Postoperative Dilatation of Superficial Temporal Artery Associated with Transient Neurologic Symptoms After Direct Bypass Surgery for Moyamoya Angiopathy

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■ **OBJECTIVE:** In moyamoya angiopathy, transient neurologic symptoms (TNS) are occasionally observed after superficial temporal artery (STA)—middle cerebral artery direct bypass surgery. The purpose of this study was to investigate the correlation between TNS and postoperative magnetic resonance imaging as well as perform a perfusion study.

■ **METHODS:** We reviewed 52 hemispheres in 33 consecutive patients with moyamoya angiopathy. TNS were defined as reversible neurologic dysfunction without any apparent intracranial infarction or hemorrhage. All patients underwent magnetic resonance imaging and single-photon emission computed tomography before and within 5 days after surgery. Maximum diameter of STA on time-of-flight magnetic resonance angiography and the dilatation ratio of STA were calculated. The presence of signal changes on fluid-attenuated inversion recovery images and regional cerebral blood flow were also evaluated.

■ **RESULTS:** TNS were observed in 13 of 52 (25%) cases 1–16 days after surgery. The mean preoperative STA dilatation, postoperative STA dilatation, and dilatation ratio of STA were $1.33 \text{ mm} \pm 0.27$, $1.67 \text{ mm} \pm 0.30$, and $29.31\% \pm 28.13\%$. Postoperative intraparenchymal cortical hyperintensity lesions and high-intensity signals in the cortex sulci (ivy sign) were detected in 24 (46.2%) cases and 29 (55.8%) cases, respectively. Univariate analyses

demonstrated no association between TNS and postoperative signal change on fluid-attenuated inversion recovery images as well as cerebral blood flow. Only >1.5-fold dilatation of STA was significantly correlated with TNS ($P < 0.0001$).

■ **CONCLUSIONS:** STA dilatation was correlated with TNS after direct bypass surgery for moyamoya angiopathy.

INTRODUCTION

Moyamoya angiopathy (MMA) is an uncommon cerebrovascular disorder characterized by progressive stenosis or occlusion of the terminal portion of the bilateral internal carotid arteries.¹ Direct bypass surgery by superficial temporal artery (STA)—middle cerebral artery (MCA) anastomosis is an established procedure^{2,3} and is considered the treatment of choice for ischemic MMA. Furthermore, a recent study revealed that direct bypass surgery for adult patients harboring hemorrhagic MMA reduced the rebleeding rate and improved the patient's prognosis.⁴ The advantage of direct bypass surgery was to obtain immediate flow improvement; however, some patients may develop transient neurologic symptoms (TNS) owing to marked changes in blood flow.^{5–8} Previous studies demonstrated that TNS were observed in 17%–61%,^{5,9–11} usually within 14 days even after successful bypass surgery, with headache, motor and/or sensory disturbance, dysarthria or

Key words

- Dilatation
- Direct bypass surgery
- Moyamoya angiopathy
- Superficial temporal artery
- Transient neurological symptom

Abbreviations and Acronyms

- CBF:** Cerebral blood flow
- CBV:** Cerebral blood volume
- CHB:** Cortical hyperintensity belt
- CHL:** Cortical hyperintensity lesion
- FLAIR:** Fluid-attenuated inversion recovery
- MCA:** Middle cerebral artery
- MMA:** Moyamoya angiopathy
- MRI:** Magnetic resonance imaging

rCC: Cerebral-to-cerebellar ratio

rSTA: Dilatation ratio of STA

STA: Superficial temporal artery

TNS: Transient neurologic symptoms

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aphasia, and epilepsy as representative symptoms. However, the pathogenic mechanism remains unclear. One study reported that patients with MMA have a significantly higher risk for symptomatic hyperperfusion as a potential complication of direct bypass surgery compared with other occlusive cerebrovascular diseases owing to the vulnerability to cerebral hyperperfusion in MMA.⁹

To date, vasogenic edema¹⁰ and local cortical hyperperfusion^{5,6,9,11} have been suggested as possible causes of TNS after direct bypass surgery. Consistent radiologic signs associated with TNS may help to diagnose and elucidate this phenomenon. The dilatation of the STA was occasionally recognized after direct bypass surgery for MMA.¹² However, the status of the STA in the acute phase of the surgery remains obscure. In this study, we investigated the correlation between TNS and postoperative magnetic resonance imaging (MRI) and performed a perfusion study based on the hypothesis that marked changes in STA diameter after bypass may be pertinent to TNS.

MATERIALS AND METHODS

Patients

All study protocols were approved by the ethics committee of Hiroshima University. The study population comprised 52 hemispheres in 33 consecutive patients with MMA who underwent STA-MCA direct bypass surgery between September 2011 and December 2016. All patients were diagnosed with MMA according to angiographic findings defined by the Japanese Research Committee on MMA. Of patients, 16 were adults or adolescents (≥ 15 years old), and 17 were children (< 15 years old). No patients had neurologic deficits before the surgery. Medical and surgical records were retrieved, and retrieved data were analyzed retrospectively. Patient demographics and characteristics are summarized in **Table 1**.

Surgical Procedure

All surgical procedures were performed by the same experienced vascular neurosurgeon (T.O.). In all cases, a parietal branch of the STA was anastomosed to a suprasylvian MCA branch (M₄). Indirect bypass by encephaloduromyosynangiosis¹³ was also performed in 19 hemispheres of 11 pediatric patients under the requirement that the brain was not relatively edematous during the surgery.

Transient Neurologic Symptoms

We reviewed all patient medical records, and TNS were defined as follows: 1) transient neurologic deficits such as motor weakness, sensory disturbance, dysarthria, or aphasia; 2) symptoms were not associated with postoperative acute cerebral infarction or intracranial hemorrhage; 3) all symptoms were resolved completely within 16 days after the surgery. Postoperative neurologic findings were evaluated at least 3 times per day. The duration of TNS was also reviewed.

Radiologic Examinations

All patients underwent computed tomography and MRI before and within 5 days of surgery. Fluid-attenuated inversion recovery (FLAIR) MRI, diffusion-weighted imaging, and time-of-flight magnetic resonance angiography were included in the MRI

Table 1. Baseline Characteristics of Study Patients

Characteristics	Value
Age, years, mean \pm SD (range)	20.4 \pm 16.6 (3–51)
Sex	
Male	6 (18.2%)
Female	27 (81.8%)
Treated hemispheres	
Adults	23 (44.2%)
Children	29 (55.8%)
Operative side	
Left	27 (51.9%)
Right	25 (48.1%)
Clinical type	
Ischemia	38 (73.1%)
Hemorrhage	4 (7.7%)
Others	10 (19.2%)

Values are presented as number (%) except for age.

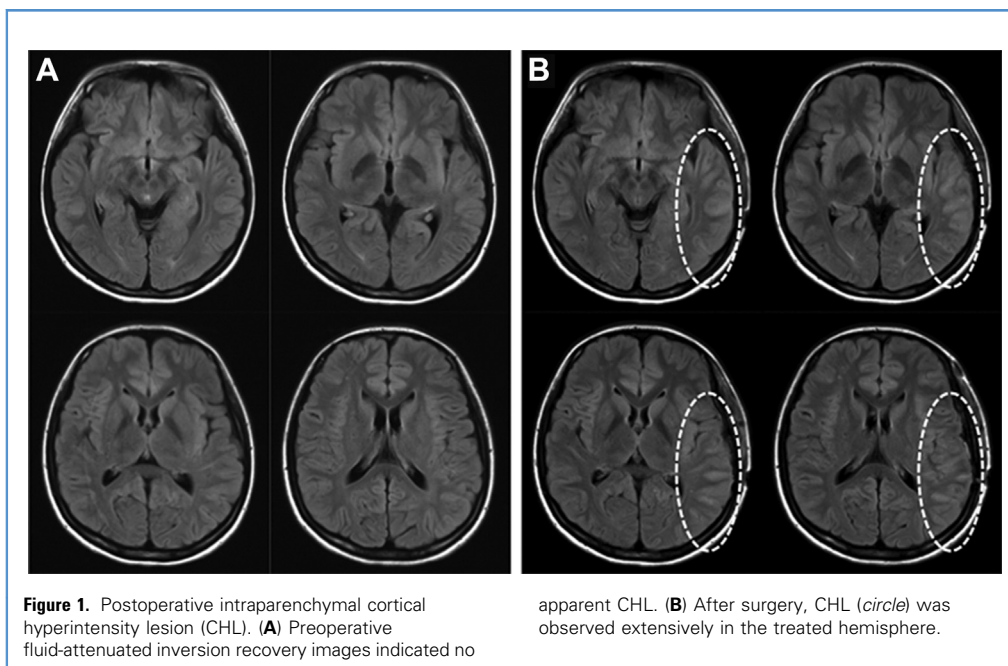
protocol. Postoperative intraparenchymal cortical hyperintensity lesion (CHL) in the treated hemisphere (**Figure 1**)¹⁰ and linear high signal intensity lesions along the cortical sulci or brain surface in the treated hemisphere (“ivy signs”) (**Figure 2**)¹⁴ were detected on FLAIR images. Both CHL and ivy signs were distinguished from acute infarction or hemorrhage by computed tomography and diffusion-weighted imaging. An increase in postoperative ivy signs was defined as de novo ivy sign.¹⁵ If TNS lasted > 5 days, MRI was performed repeatedly to investigate the occurrence of cerebral infarction or hemorrhage.

Dilatation Ratio of STA

The maximum diameter of the STA was measured at the straight section of the parietal branch on magnetic resonance angiography. Images were analyzed by 2 experienced vascular neurosurgeons (S.S. and T.M.) who were blinded to the neurologic condition and outcome of each case in a consensus reading using an open-source medical image viewer (Horos; <http://www.horosproject.org/>). Maximum intensity projections were reconstructed in axial, coronal, and sagittal views to identify running courses of the major trunk of the STA. Along the running course of the STA, longitudinal and perpendicular maximum intensity projection views (slab thickness of 2.2 mm) were reconstructed, and the maximum diameter of the STA was measured (**Figure 3**). The dilatation ratio of STA (rSTA) was calculated with the following formula: $rSTA(\%) = (STA_{post} - STA_{pre}) / STA_{pre} \times 100$.

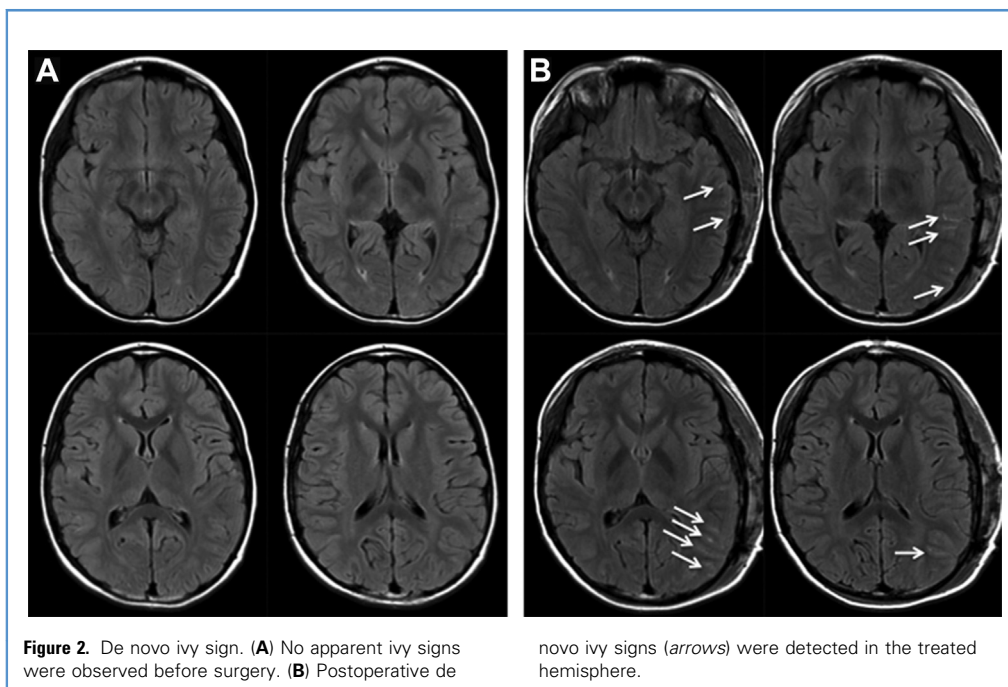
Cerebral Blood Flow Measurement

Single-photon emission computed tomography using iodine-123-N-isopropyl-p-iodoamphetamine was performed in all patients before and within 3 days after the surgery. All cerebral blood flow (CBF) images were transformed into the standard brain size and



shape by linear and nonlinear parameters with the system for anatomic standardization developed by Minoshima et al.^{16,17} The region of interest was selected in bilateral MCA territories on both axial levels of the basal ganglia (MCA 1), the centrum semiovale (MCA 2), and bilateral cerebellum using NEURO FLEXER software (Nihon MediPhysics Co., Ltd., Tokyo, Japan).¹⁸ Regional CBF was

evaluated using semiquantitative parameters calculated from mean single-photon emission computed tomography counts of the MCA territories and cerebellum, respectively. The cerebral-to-cerebellar ratio (rCC) was defined as the ratio of resting single-photon emission computed tomography counts in regions of interest of the treated MCA territories to the ipsilateral



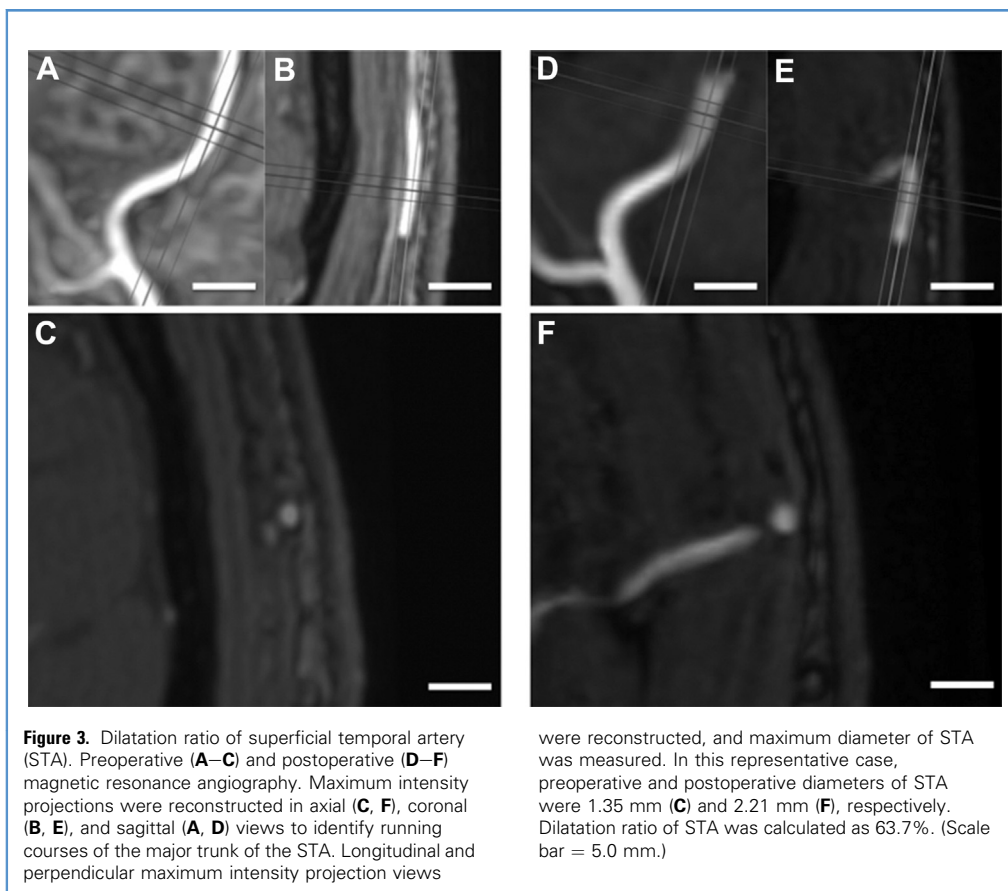


Figure 3. Dilatation ratio of superficial temporal artery (STA). Preoperative (A–C) and postoperative (D–F) magnetic resonance angiography. Maximum intensity projections were reconstructed in axial (C, F), coronal (B, E), and sagittal (A, D) views to identify running courses of the major trunk of the STA. Longitudinal and perpendicular maximum intensity projection views

were reconstructed, and maximum diameter of STA was measured. In this representative case, preoperative and postoperative diameters of STA were 1.35 mm (C) and 2.21 mm (F), respectively. Dilatation ratio of STA was calculated as 63.7%. (Scale bar = 5.0 mm.)

cerebellum. Preoperative patient hemodynamics were evaluated by rCC. The increasing rCC was calculated as the ratio of postoperative to preoperative rCC.

Statistical Analyses

All statistical analyses were performed using JMP version 10.0 (SAS Institute Inc., Cary, North Carolina, USA). Values are presented as mean \pm SD. Categorical variables were compared by the Fisher exact probability test. Continuous variables with normal distributions were analyzed by Student t test and variables with nonnormal distributions were analyzed by Mann-Whitney U test. The incidence of TNS was calculated, and the correlation of age, sex, side of hemisphere, operative procedure, type of preoperative symptom, rSTA, CHL, de novo ivy sign, preoperative rCC, and increasing rCC with TNS was evaluated using univariate analyses. Significance was defined as a P value < 0.05 .

RESULTS

Successful patency of the direct bypass, which was assessed by intraoperative indocyanine green angiography and postoperative magnetic resonance angiography, was obtained in all patients. No stenosis at the anastomosis was seen in any of the cases. No symptomatic hemorrhagic or ischemic complications were observed.

Frequency and Variety of TNS

TNS were observed in 13 (25%) cases 1–16 days after the surgery, lasting 7.3 days ± 4.0 . Among all TNS, signs and symptoms observed included motor weakness in 5 (9.6%) cases, sensory disturbance in 8 (15.4%), dysarthria in 2 (3.8%), and aphasia in 7 (13.5%).

Dilatation of STA

No shrinkage of the anastomosed STA was observed after surgery. The mean maximum diameter of the preoperative and postoperative STA was 1.33 mm ± 0.27 and 1.67 mm ± 0.30 , respectively. Based on these values, the mean rSTA was calculated as $29.31\% \pm 28.13$. There was no significant correlation between indirect bypass procedure and rSTA ($P = 0.92$).

Signal Changes on FLAIR Images

No apparent CHL was found before surgery. Preoperative ivy sign was observed in 16 hemispheres in 10 (19.2%) patients. Postoperative CHL and de novo ivy sign in the treated hemisphere were detected in 24 (46.2%) and 29 (55.8%) cases, respectively.

Postoperative CBF Increase

The mean preoperative rCC at MCA 1 and 2 was 0.97 ± 0.17 and 0.95 ± 0.17 , respectively. The mean increasing rCC at MCA 1 and

Table 2. Factors Associated with Transient Neurologic Symptoms

Variable	OR	95% CI		P Value
		Lower	Upper	
Age	0.34	0.06	1.87	0.21
Sex (female)	1.24	0.30	6.35	0.78
Side (left)	2.75	0.82	10.27	0.10
Operative procedure (indirect bypass)	0.37	0.09	1.30	0.12
Preoperative symptom (ischemia)	1.91	0.49	9.52	0.36
rSTA (>50%)	28.33	5.80	218.74	<0.0001*
CHL	2.62	0.79	9.27	0.11
De novo ivy sign	2.2	0.66	8.19	0.20
Preoperative rCC				
MCA 1	3.08	0.18	63.48	0.44
MCA 2	2.31	0.16	39.24	0.54
Increasing rCC				
MCA 1	0.42	0.02	10.87	0.59
MCA 2	0.65	0.03	15.93	0.78

CI, confidence interval; OR, odds ratio; rSTA, dilatation ratio of superficial temporal artery; CHL, cortical hyperintensity lesion; rCC, cerebral-to-cerebellar ratio; MCA, middle cerebral artery.
*Significant.

2 was 0.99 ± 0.11 and 1.02 ± 0.14 , respectively. The increasing rCC was >1.0 at MCA 1 and MCA 2 in 20 (38.5%) cases and 24 (46.2%) cases, respectively.

Factors Associated with TNS

Univariate analyses are summarized in Table 2. No significant correlation was found between TNS and patient demographics such as age, sex, side of hemisphere, operative procedure, or type of preoperative symptom. The radiographic variables, including CHL, de novo ivy sign, preoperative rCC, and increasing rCC, also had no significant association with TNS. However, rSTA was significantly correlated with TNS ($P < 0.0001$).

DISCUSSION

This is the first report to our knowledge to demonstrate a correlation between TNS and marked dilatation of the STA after direct bypass surgery for MMA. No stenosis of the anastomosis was observed; therefore, it was unnecessary to consider if poor anastomosis contributed to dilatation of the STA. Among possible predictors for TNS, rSTA $>50\%$ was significantly correlated with TNS. rSTA as a continuous variable suggested that a 1% rise in rSTA would increase the risk of TNS by 7% (odds ratio 1.07, 95% confidence interval 1.04–1.12, $P < 0.0001$). Direct bypass surgery for MMA has been established; however, the relatively high incidence of TNS of 17%–61.0%^{5,9-11} is of concern. One study

reported that patients with MMA have a significantly higher risk for symptomatic hyperperfusion as a potential complication of direct bypass surgery compared with other occlusive cerebrovascular diseases owing to the vulnerability to cerebral hyperperfusion in MMA.⁹

The precise mechanism of TNS and its associated radiographic findings remain controversial. Studies have suggested vasogenic edema¹⁰ and local cortical hyperperfusion^{5,6,9,11} as causes of TNS, indicating some characteristic findings associated with these hypotheses. Moreover, transcranial Doppler ultrasound was reported to provide clinical data on the hemodynamics in MMA before and after revascularization.¹⁹ However, it was not evaluated in this study because of the necessity of sedation in most pediatric cases.

The appearance of a cortical hyperintensity belt (CHB) sign, which was defined as the presence of an intraparenchymal high-intensity signal on FLAIR images after direct bypass surgery for MMA, was demonstrated to be significantly correlated with TNS.¹⁰ It was speculated that the CHB sign was due to vasogenic edema, and it was hypothesized that preoperative vasodilatation and hemodynamic changes caused by direct revascularization may lead to autoregulatory failure and extravasation of fluid.¹⁰ In this study, CHL was defined in a similar manner with the CHB sign. Furthermore, all signs disappeared by 1 month after surgery, and no high signal intensity on diffusion-weighted imaging was observed, which was consistent with the signal trait of CHB signs. However, CHL was not significantly correlated with TNS. Thus, CHL itself may not be the cause of TNS, but rather may result from extravasation owing to autoregulatory failure, or the incidence of CHL may depend on the extent of vulnerability of the blood-brain barrier. The expression of vascular endothelial growth factor²⁰ and matrix metalloproteinase 9,²¹ both of which affect the permeability of the blood-brain barrier, was significantly increased in patients with MMA compared with healthy control subjects. Therefore, vasogenic edema may be observed frequently after direct bypass surgery for MMA. Consequently, CHL may be observed occasionally with or without TNS.

A de novo ivy sign indicated a focal increase in CBF in pial vessels in MMA and was suggested as an independent factor related to postoperative symptomatic hyperperfusion.¹⁵ In this study, preoperative rCC was not correlated with TNS. Recently, preoperative CBF and cerebrovascular reserve were found not to predict postoperative symptomatic hyperperfusion in MMA, which was consistent with our results.^{22,23} However, increasing rCC, which may be associated with hyperperfusion, and the de novo ivy sign were not correlated with TNS.

Regarding hemodynamics, studies have revealed that symptomatic hyperperfusion in MMA was characterized by temporary increases in CBF $>100\%$ over the preoperative value caused by prolonged recovery of increased cerebral blood volume (CBV).²² Preoperative CBV increase may be an independent predictor of both radiologic and symptomatic hyperperfusion after surgery in adult MMA.²³ These reports suggested that TNS was, at least in part, correlated with increasing CBV before and after direct bypass surgery for MMA. Using intraoperative indocyanine green angiography, Awano et al.²⁴ reported that the perfusion pressure in MMA was lower than in non-MMA, which may cause a larger pressure gradient between the anastomosed STA and recipient vessels. Theoretically, an increased CBV suggests autoregulatory

vasodilatation in response to the cerebral perfusion pressure reduction.²⁵ It is speculated autoregulatory failure may occur in MMA; cortical arteries may be dilated, and cerebral perfusion pressure may be reduced before the surgery. Consequently, excessive increase in CBV may occur immediately after direct bypass surgery, which may induce TNS secondary to a metabolic disorder. Simultaneously, the marked dilatation of the STA may be induced with TNS as a result of a large pressure gradient between the graft and recipient vessel.

In the present study, the increasing rCC of MCA 1 and 2 was >1.0 (100%) in 20 (38.5%) and 24 (46.2%) cases, respectively. This suggested that “radiographic” hyperperfusion frequently occurs after direct bypass surgery for MMA; however, the incidence of “symptomatic” hyperperfusion may be relatively low. It has been reported that the CBF increased ratio with or without TNS was not significantly different between these 2 groups.¹⁰ Therefore, it seems controversial to attribute all causes of TNS to local hyperperfusion. Both CHL and local hyperperfusion are likely minimally related to TNS; however, rSTA may have a high sensitivity to TNS. Based on our results, we hypothesize that increasing CBV may be a primary cause of TNS, and rSTA may be a useful predictor of TNS after direct bypass surgery for MMA. If the diameter of the anastomosed STA increased >50%, blood pressure should be strictly controlled to avoid postoperative cerebral hemorrhage and/or epilepsy, and neurologic data should be evaluated frequently.

This study has some limitations. First, this was a retrospective cohort study in a single center with a small sample size. For further evaluation, a prospective study involving a greater number of patients and multivariate analysis may be needed for confirmation of these initial results. Second, CBF was evaluated using semiquantitative parameters. It was difficult to evaluate CBF quantitatively in children because of the necessity of arterial blood sampling. This lack of a highly quantitative evaluation regarding CBF may have contributed to the irrelevance between increasing rCC and TNS. Moreover, CBF was evaluated just 1 time within 3 days after surgery. Therefore, it is possible that CBF may further increase in the other acute phase after surgery. Third, an increase in CBV was not confirmed. CBV needs to be evaluated before and after surgery to validate our hypothesis described here.

CONCLUSIONS

In this retrospective study, rSTA was correlated with TNS after direct bypass surgery for MMA. The occurrence of TNS should be considered in patients whose anastomosed STA diameter increases >50%.

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REFERENCES

- Suzuki J, Takaku A. Cerebrovascular “moyamoya” disease. Disease showing abnormal net-like vessels in base of brain. *Arch Neurol.* 1969;20:288-299.
- Karasawa J, Touho H, Ohnishi H, Miyamoto S, Kikuchi H. Long-term follow-up study after extracranial-intracranial bypass surgery for anterior circulation ischemia in childhood moyamoya disease. *J Neurosurg.* 1992;77:84-89.
- Miyamoto S, Akiyama Y, Nagata I, Karasawa J, Nozaki K, Hashimoto N, et al. Long-term outcome after STA-MCA anastomosis for moyamoya disease. *Neurosurg Focus.* 1998;5:e5.
- Miyamoto S, Yoshimoto T, Hashimoto N, Okada Y, Tsuji I, Tominaga T, et al. Effects of extracranial-intracranial bypass for patients with hemorrhagic moyamoya disease: results of the Japan Adult Moyamoya Trial. *Stroke.* 2014;45:1415-1421.
- Fujimura M, Kaneta T, Mugikura S, Shimizu H, Tominaga T. Temporary neurologic deterioration due to cerebral hyperperfusion after superficial temporal artery-middle cerebral artery anastomosis in patients with adult-onset moyamoya disease. *Surg Neurol.* 2007;67:273-282.
- Ogasawara K, Komoribayashi N, Kobayashi M, Fukuda T, Inoue T, Yamadate K, et al. Neural damage caused by cerebral hyperperfusion after arterial bypass surgery in a patient with moyamoya disease: case report. *Neurosurgery.* 2005;56:E1380 [discussion: E1380].
- Sasamori T, Kuroda S, Nakayama N, Iwasaki Y. Incidence and pathogenesis of transient cheiro-oral syndrome after surgical revascularization for moyamoya disease. *Neurosurgery.* 2010;67:1054-1059 [discussion: 1060].
- 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).* 1998;38:420-424.
- Fujimura M, Shimizu H, Inoue T, Mugikura S, Saito A, Tominaga T. Significance of focal 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]I)iodoamphetamine single-photon emission computed tomography. *Neurosurgery.* 2011;68:957-964 [discussion: 964-965].
- Hamano E, Kataoka H, Morita N, Maruyama D, Satow T, Iihara K, et al. Clinical implications of the cortical hyperintensity belt sign in fluid-attenuated inversion recovery images after bypass surgery for moyamoya disease. *J Neurosurg.* 2017;126:1-7.
- 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 neurological deterioration. *Cerebrovasc Dis.* 2008;25:580-586.
- Honda M, Kitagawa N, Tsutsumi K, Morikawa M, Nagata I, Kaminogo M. Magnetic resonance angiography evaluation of external carotid artery tributaries in moyamoya disease. *Surg Neurol.* 2005;64:325-330.
- Karasawa J, Kikuchi H, Furuse S, Sakaki T, Yoshida Y. A surgical treatment of “moyamoya” disease “encephalo-myo synangiosis.”. *Neurol Med Chir (Tokyo).* 1977;17:29-37.
- Maeda M, Tsuchida C. “Ivy sign” on fluid-attenuated inversion-recovery images in childhood moyamoya disease. *AJNR Am J Neuroradiol.* 1999;20:1836-1838.
- Horie T, Morikawa M, Morofuji Y, Hiu T, Izumo T, Hayashi K, et al. De novo ivy sign indicates postoperative hyperperfusion in moyamoya disease. *Stroke.* 2014;45:1488-1491.
- Minoshima S, Koeppe RA, Frey KA, Kuhl DE. Anatomic standardization: linear scaling and nonlinear warping of functional brain images. *J Nucl Med.* 1994;35:1528-1537.
- Senda M, Ishii K, Oda K, Sadato N, Kawashima R, Sugiura M, et al. Influence of ANOVA design and anatomical standardization on statistical mapping for PET activation. *Neuroimage.* 1998;8:283-301.
- Ogura T, Hida K, Masuzuka T, Saito H, Minoshima S, Nishikawa K. An automated ROI setting method using NEUROSTAT on cerebral blood flow SPECT images. *Ann Nucl Med.* 2009;23:33-41.

19. Cho H, Jo KI, Yu J, Yeon JY, Hong SC, Kim JS. Low flow velocity in the middle cerebral artery predicting infarction after bypass surgery in adult moyamoya disease. *J Neurosurg.* 2017;126:1573-1577.
20. Sakamoto S, Kiura Y, Yamasaki F, Shibukawa M, Ohba S, Shrestha P, et al. Expression of vascular endothelial growth factor in dura mater of patients with moyamoya disease. *Neurosurg Rev.* 2008;31:77-81 [discussion: 81].
21. Fujimura M, Watanabe M, Narisawa A, Shimizu H, Tominaga T. Increased expression of serum matrix metalloproteinase-9 in patients with moyamoya disease. *Surg Neurol.* 2009;72:476-480 [discussion: 480].
22. Kaku Y, Iihara K, Nakajima N, Kataoka H, Fukuda K, Masuoka J, et al. Cerebral blood flow and metabolism of hyperperfusion after cerebral revascularization in patients with moyamoya disease. *J Cereb Blood Flow Metab.* 2012;32:2066-2075.
23. Uchino H, Kuroda S, Hirata K, Shiga T, Houkin K, Tamaki N. Predictors and clinical features of postoperative hyperperfusion after surgical revascularization for moyamoya disease: a serial single photon emission CT/positron emission tomography study. *Stroke.* 2012;43:2610-2616.
24. Awano T, Sakatani K, Yokose N, Kondo Y, Igarashi T, Hoshino T, et al. Intraoperative EC-IC bypass blood flow assessment with indocyanine green angiography in moyamoya and non-moyamoya ischemic stroke. *World Neurosurg.* 2010;73:668-674.
25. Powers WJ, Grubb RL Jr, Raichle ME. Physiological responses to focal cerebral ischemia in humans. *Ann Neurol.* 1984;16:546-552.

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