Magnetic Resonance Imaging/Gadolinium-enhanced Vessel Wall Image after Bypass Surgery in Moyamoya Disease

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Objective: After bypass surgery in patients with moyamoya disease, several changes on magnetic resonance imaging (MRI)/fluid attenuated inversion recovery (FLAIR) have been recognized, while findings on MRI/gadolinium-enhanced (Gd) vessel wall imaging (VWI) have never been reported. The purposes of this study were to investigate postoperative changes on MRI/Gd VWI and to clarify the relationship between the MRI/Gd VWI and MRI/FLAIR findings.
Methods: Consecutive patients who underwent bypass surgery at our hospital from September 2020 to March 2022 were candidates.
Results: In 20 patients with moyamoya disease, 25 operated hemispheres were investigated. In all hemispheres, hyperintensities in the cortical sulci on MRI/FLAIR and enhancement in the cortical sulci on MRI/Gd VWI appeared after bypass surgery. The maximum appearance of sulci enhancement on MRI/Gd VWI occurred earlier than the maximum appearance of the sulci hyperintensity on MRI/FLAIR, and this difference was significant ($p = 0.001$).
Conclusions: MRI/Gd VWI demonstrated that the peak of the enhancement changes preceded the peak of hyperintensity changes on MRI/FLAIR. These MRI changes may reflect alterations in blood-brain barrier permeability after bypass surgery in patients with moyamoya disease.

Key words: bypass, complication, hyperperfusion, magnetic resonance image, moyamoya disease

INTRODUCTION

In patients with moyamoya disease, magnetic resonance imaging (MRI)/fluid attenuated inversion recovery (FLAIR) frequently shows hyperintensity in the cortical sulci, which is termed as the "ivy sign [1]." After bypass surgery, further hyperintensity changes occur in both cerebral sulci and the cerebral cortex on MRI/FLAIR, and cerebral blood flow alterations after surgery are suspected to cause these changes [2]. Recently, the presence of arterial wall enhancement in major arteries has been reported on MRI/gadolinium-enhanced (Gd) vessel wall imaging (VWI) in conservatively treated patients with moyamoya disease, which could be related to disease progression [3], while no study has reported MRI/Gd VWI findings in the early phase after bypass surgery. As we routinely perform MRI perfusion using Gd after bypass surgery to evaluate hyperperfusion, MRI/VWI was performed immediately after MRI perfusion. We then found enhancement in the cerebral sulci on the MRI/Gd VWI, which appeared in areas similar to where sulci hyperintensity was found on MRI/FLAIR. The cortical surface above the cortical hyperintensity observed on MRI/FLAIR was also enhanced on MRI/Gd VWI.

In this study, we evaluated the relationship between sulci hyperintensity on MRI/FLAIR and sulci enhancement on MRI/Gd VWI after bypass surgery. Then, we investigated the relationship between cortical hyperintensity on MRI/FLAIR and surface enhancement on MRI/Gd VWI.

MATERIAL AND METHODS

Patient population

We retrospectively evaluated patients with moyamoya disease who underwent bypass surgery between September 2020 and March 2022. All patients were diagnosed with moyamoya disease according to the Guidelines for Diagnosis and Treatment of Moyamoya Disease [4]. Patients who underwent MRI/FLAIR and MRI/Gd VWI before and after surgery were included, while patients younger than 16 years were excluded. This study was approved by the Tokai University Ethics Committee (22R076).

Clinical evaluations

The following patient information was collected from the medical records: age, sex, family history of moyamoya disease, mode of onset, preoperative modified Rankin Scale (mRS) score, whether the moyamoya disease was bilateral or unilateral, first or second surgery, presence or absence of postoperative hyperperfusion syndrome, and mRS score 3 months after surgery. All patients underwent superficial temporal artery (STA)–middle cerebral artery (MCA) double bypass, in which both the frontal and parietal branches of the

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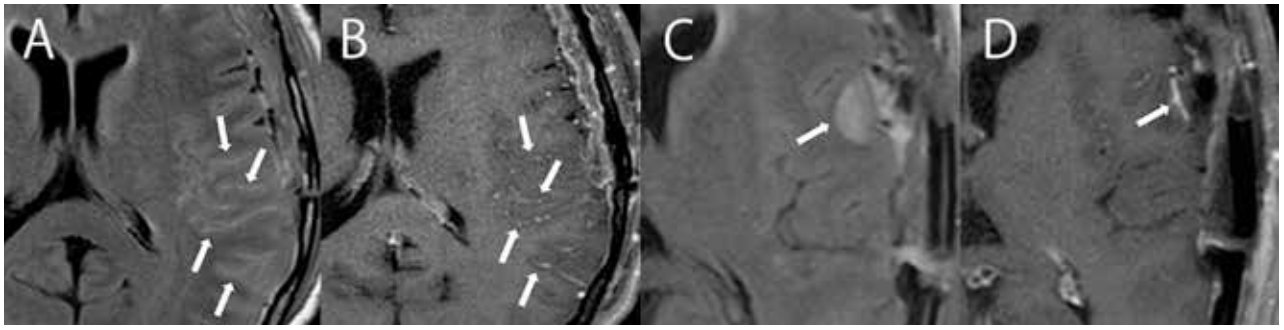

Fig. 1 Postoperative changes on magnetic resonance imaging (MRI)/fluid attenuation inversion recovery (FLAIR) (A, C) and MRI/gadolinium-enhanced (Gd) vessel wall imaging (VWI) (B, D). A, B: Postoperative changes in the cortical sulci. As MRI/FLAIR showed sulci hyperintensity (A), MRI/Gd VWI revealed sulci enhancement in almost the same area (B). C, D: Postoperative changes in the cerebral cortex and its surface. Enhancement of the cortical surface is demonstrated on MRI/Gd VWI (D) just above a cortical hyperintensity area on MRI/FLAIR (C). The white arrow indicates postoperative changes.

STA were anastomosed with the M4 segments of the MCA associated with encephalo-duro-myo-synangiosis. Postoperative hyperperfusion syndrome was defined as the occurrence of paresis, sensory impairment, speech disorder, and/or headache associated with focal hyperperfusion on single-photon emission computed tomography (SPECT) [5].

Image acquisition

MRI was performed using a 1.5-T MR scanner (Ingenia 1.5T, PHILIPS, Amsterdam Nederland). Diffusion-weighted imaging, magnetic resonance angiography (MRA), T1-weighted imaging (WI), T2-WI, FLAIR, VWI (T1-volume isotropic turbo spin-echo acquisition (VISTA)), perfusion MRI, and Gd VWI were performed in that order. FLAIR and VISTA were performed under the following conditions: FLAIR: TR/TE = 6000/120, IR delay = 2000, FOV = 240 × 193 mm^2, matrix size = 0.94 × 0.94, slice gap = 1, slice thickness = 5, time = 2 min 36 sec. VISTA T1Gd (same as the plain): TR/TE = 400/31, FOV = 200 × 200 mm^2, matrix size = 0.42 × 0.42 × 0.7, slice gap = 0, slice thickness = 0.7, time = 3 min 25 sec. In principle, MRI was performed preoperatively at 1, 3, 6, 13, and 20 days and 1, 3, and 6 months after surgery. SPECT with N-isopropyl-(I^{123})-p-iodoamphetamine (IMP) using a scanner (Symbia Evo, Siemens, Germany) was performed 1, 6, and 13 days after surgery.

Imaging analysis/interpretation

Postoperative hyperintensity changes on MRI/FLAIR were observed in the sulci and the cerebral cortex. The former was termed sulci hyperintensity (Fig. 1A), while the latter was termed cortical hyperintensity (Fig. 1C). The postoperative enhancement changes on MRI/Gd VWI were also observed in the sulci and cortical surface. The former was termed sulci enhancement (Fig. 1B), while the latter was termed surface enhancement (Fig. 1D). The Alberta Stroke Program Early CT Score (ASPECTS) was used for segmentation [6]. In addition to the M1, M2, M3, M4, M5, and M6 regions according to ASPECTS, a pre-M1 segment frontal to the M1 segment and a post-M3 segment occipital to the M3 segment were registered as modified ASPECTS. Eight regions in each patient were evaluated according to the method described below. Sulci hyperintensity in each area on MRI/FLAIR was evaluated as follows [7]: Grade 0, no sulci hyperintensity; Grade 1 represents the sulci hyperintensity in less than half of the region; Grade 2 represents the sulci hyperintensity in more than half of the region. The total value was expressed as the sulci hyperintensity score. Sulci enhancement in each area on MRI/Gd VWI was defined as follows: Grade 0, no sulci enhancement; Grade 1 represents the sulci enhancement that was less than half of the region; Grade 2 represents the sulci enhancement that was more than half of the region.

Table 1 Characteristics of 25 operated hemispheres in 20 patients undergoing bypass surgery for moyamoya disease.

Age (years, mean ± standard deviation)	39.9 ± 14.3
Female	19 (76%)
Family history	8 (32%)
Onset	
*TIA	10 (40%)
Ischemia	8 (32%)
Hemorrhage	5 (20%)
Others	2 (8%)
Preoperative †mRS 0–1	23 (92%)
Bilateral moyamoya disease	23 (92%)
Left hemisphere	11 (44%)
First surgery	12 (48%)
Suzuki stage	
2	4 (16%)
3	10 (40%)
4	9 (36%)
5	2 (8%)
‡CBF < 80%	10 (40%)
Impaired §CVR (< 10%)	14 (56%)
Postoperative hyperperfusion syndrome	16 (64%)
mRS 0–1, three months after surgery	23 (92%)

*TIA = transient ischemic attack; †mRS = modified Rankin Scale; ‡CBF = cerebral blood flow; §CVR = cerebrovascular reserve

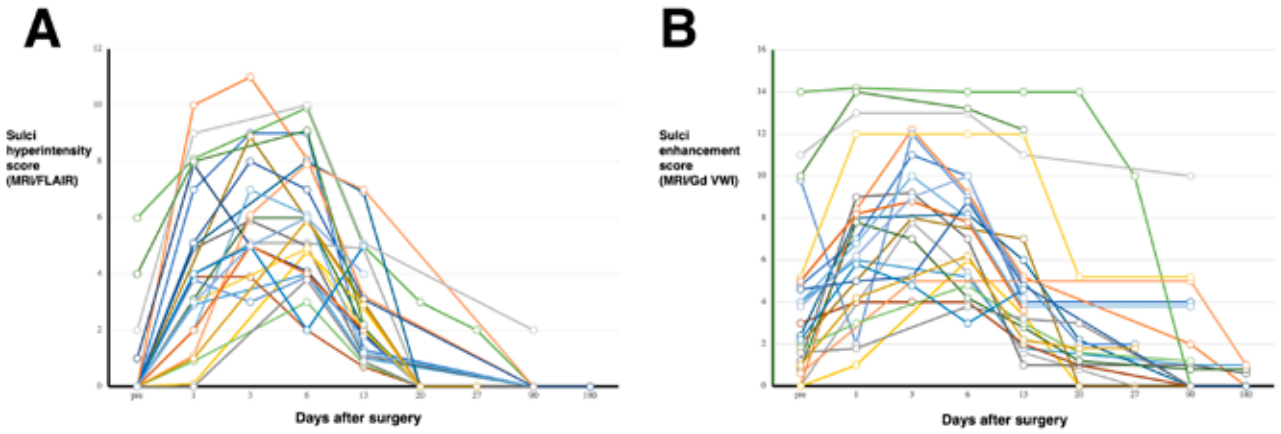

Fig. 2 Time courses of the sulci hyperintensity score on magnetic resonance imaging (MRI)/fluid-attenuated inversion recovery (FLAIR) (A), the sulci enhancement score on MRI/gadolinium-enhanced (Gd) vessel wall imaging (VWI) (B). A: The peak value of the sulci hyperintensity score was observed 4.8 ± 1.6 (mean ± standard deviation) days after surgery. B: The peak value of the sulci enhancement score was observed 2.9 ± 2.0 days after surgery. The colors indicate each operated hemisphere.

The total value was defined as the sulci enhancement score.

The maximum diameter of cortical hyperintensity was measured on MRI/FLAIR. The intensity of surface enhancement in each area on MRI/Gd VWI was defined as follows: Grade 0, no enhancement; Grade 1, mild enhancement, where the intensity of sulci enhancement was less than that of the pituitary infundibulum; Grade 2, strong enhancement, where the intensity of sulci enhancement was similar to or higher than that of the infundibulum [8]. The total value was expressed as the surface enhancement intensity score. The Suzuki stage of moyamoya disease was assessed using preoperative cerebral angiography. Cerebral blood flow (CBF) was semi-quantitatively evaluated using the Patlak plot method in SPECT images. Preoperative hypoperfusion was defined as CBF less than 80% of the normal value. Cerebrovascular reserve capacity impairment was defined as an increase in blood flow less than 10% after acetazolamide loading. CBF in each region of the modified ASPECTS and cerebellar hemispheres was measured in a region of interest (ROI) 1 cm in diameter. CBF in each area was divided by the cerebellar CBF value on the same side. The postoperative increase ratio in CBF was calculated as the value of the postoperative CBF divided by the preoperative CBF. All images were independently evaluated by two neurosurgeons (T.S. and T.Y.). After the independent review, a consensus reading was achieved to resolve any differences in interpretation.

Statistical analysis

Data were expressed as the mean ± standard deviation. Chi square test or Fisher's exact probability test was used to evaluate categorical variables. Wilcoxon's signed-rank test was used to compare two total numbers of categorical values, while the Mann-Whitney U test was used to compare continuous variables. Pearson's correlation coefficient was used to evaluate the relationship between continuous variables. Statistical analyses were performed using SPSS for Windows version 26 (IBM, Chicago, IL, USA). P values < 0.05 were considered significant.

RESULTS

Between September 2020 and March 2022, 21 consecutive patients with moyamoya disease in 26 hemispheres underwent bypass surgery. One patient with a gadolinium allergy was excluded. All the patients were 16 years of age or older. Therefore, 25 operated hemispheres in 20 patients were evaluated in this study. Patient characteristics are listed in Table 1. Patency of the bypass in all patients was confirmed on postoperative MRA. No major postoperative complications other than hyperperfusion syndrome occurred in all 25 surgeries.

Sulci hyperintensity on MRI/FLAIR and sulci enhancement on MRI/Gd VWI

Both sulci hyperintensity on MRI/FLAIR and sulci enhancement on MRI/Gd VWI were observed in all 25 hemispheres (100%). On the contrary, no postoperative change in the contralateral hemisphere was demonstrated by either MRI/FLAIR or MRI/Gd VWI. Fig. 2 shows the time courses of the sulci hyperintensity score on MRI/FLAIR and the sulci enhancement score on MRI/Gd VWI. The peak values of the sulci hyperintensity score (Fig. 2A) and the sulci enhancement score (Fig. 2B) were observed at 4.8 ± 1.6 and 2.9 ± 2.0 days after surgery, respectively. The peak of the sulci enhancement score on MRI/Gd VWI occurred significantly earlier than the peak of the sulci hyperintensity score on MRI/FLAIR ($p = 0.001$). Preoperative sulci hyperintensity on MRI/FLAIR was observed in 4 hemispheres, while sulci enhancement was observed by MRI/Gd VWI in 22 hemispheres. In 20 of the 25 hemispheres, MRI was performed more than 3 months after surgery. The sulci hyperintensity score decreased to the preoperative value at 46.5 ± 35.8 days after surgery. An improvement in the sulci enhancement score to the preoperative values occurred 35.4 ± 51.1 days after surgery. In 10 of the 20 hemispheres, sulci enhancement remained 3 months after surgery. A correlation between the maximum hyperintensity score on MRI/FLAIR and the maximum sulci enhancement score on MRI/Gd VWI is shown in Fig. 3. Correlation analysis revealed a statistically significant relationship

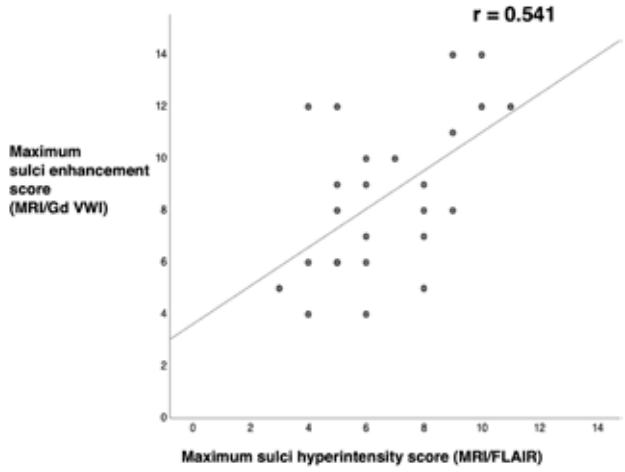

Fig. 3 A relationship between the maximum sulci hyperintensity score on magnetic resonance imaging (MRI)/fluid-attenuated inversion recovery (FLAIR) and the maximum sulci enhancement score on MRI/gadolinium-enhanced (Gd) vessel wall imaging (VWI), which show a significant correlation (p = 0.005). A black circle indicates each operated hemisphere. A line indicates a regression line.

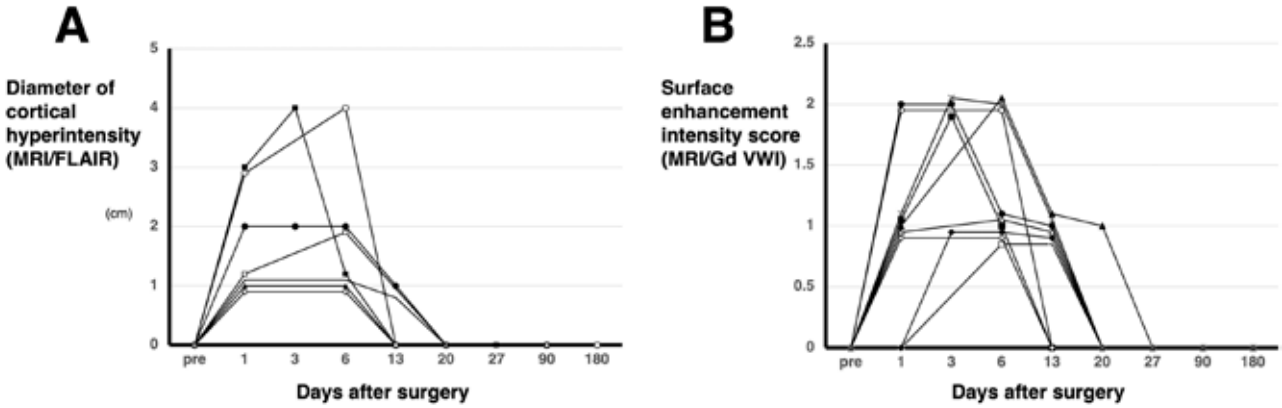

Fig. 4 Time courses of a diameter of cortical hyperintensity on magnetic resonance imaging (MRI)/fluid-attenuated inversion recovery (FLAIR) (A) and the surface enhancement intensity score on MRI/gadolinium-enhanced (Gd) vessel wall imaging (VWI) (B). A: The maximum diameter of cortical hyperintensity was observed 5.14 ± 1.46 (mean ± standard deviation) days after surgery, and the cortical hyperintensity disappeared 16.0 ± 3.74 days after surgery. B: The peak of the surface enhancement intensity score was observed 2.71 ± 2.36 days after surgery and the surface enhancement disappeared 24.2 ± 25.8 days after surgery. Each symbol indicates each operated hemisphere.

between the maximum sulci hyperintensity score and the maximum sulci enhancement score (r = 0.541, p = 0.005).

Cortical hyperintensity on MRI/FLAIR and surface enhancement on MRI/Gd VWI

Cortical hyperintensity on MRI/FLAIR was observed in 7 of the 25 hemispheres after surgery. In all 7 hemispheres, the brain surface above the cortical hyperintensity was enhanced by contrast medium. Surface enhancement was shown in 9 hemispheres, including 2 hemispheres without FLAIR cortical hyperintensity. Above the cortical hyperintensity on MRI/FLAIR, surface enhancement was frequently and significantly observed compared with hemispheres without cortical hyperintensity (p = 0.001). Fig. 4 shows the time courses of the diameter of cortical hyperintensity on MRI/FLAIR and the surface enhancement intensity score on MRI/Gd VWI. The maximum diameter of cortical hyperintensity and the maximum surface enhancement intensity score were observed at 5.14 ± 1.46 and 2.71 ± 2.36 days after surgery, respectively. The cortical hyperintensity and the surface enhancement disappeared at 16.0 ± 3.74 and 24.2 ± 25.8 after surgery, respectively.

Relationship between postoperative hyperperfusion syndrome and imaging findings

Postoperative hyperperfusion syndrome was observed after 16 of 25 surgeries. Patients with postoperative hyperperfusion syndrome showed significantly higher sulci enhancement scores compared with patients without hyperperfusion syndrome (p = 0.01). A significantly larger CBF increase on postoperative SPECT was found in patients with postoperative hyperperfusion syndrome compared with patients without hyperperfusion syndrome (p = 0.002). Hyperperfusion syndrome was observed in 3 of 7 hemispheres (42.9%) with cortical hyperintensity and in 13 of 18 hemispheres (72.2%) without cortical hyperintensity. The occurrence of hyperperfusion syndrome was not different significantly between the hemispheres with and without cortical hyperintensity (p = 0.21).

An illustrative case

A 54-year-old female with moyamoya disease, who presented with ischemia-related symptoms, underwent left bypass surgery (Fig. 5). Preoperative MRI/FLAIR did not show any sulci hyperintensity (Fig. 5A), but preoperative MRI/Gd VWI demonstrated a small area of sulci enhancement in the left hemisphere (Fig. 5B). Although several hyperintense lesions in the sulci ap-

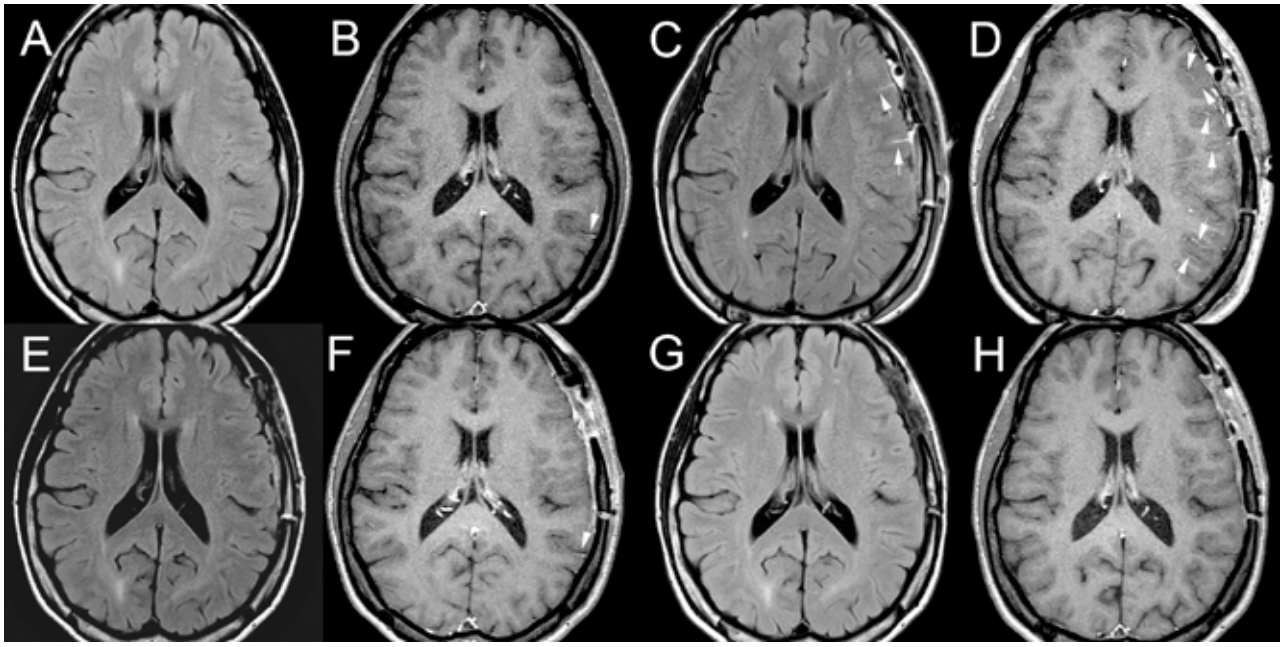

Fig. 5 Postoperative changes on magnetic resonance imaging (MRI)/fluid attenuation inversion recovery (FLAIR) (A, C, E, G) and MRI/gadolinium-enhanced (Gd) vessel wall imaging (VWI) (B, D, F, H) in a 54-year-old female with moyamoya disease who underwent left bypass surgery. A, B: Preoperative MRI. MRI/FLAIR shows no apparent sulci hyperintensity (A), while MRI/Gd VWI demonstrates linear enhancement in the left temporal sulcus (B). C, D: MRI 3 days after surgery. MRI/FLAIR shows hyperintense lesions in the sulci of the left frontal lobe (C). MRI/Gd VWI reveals linear enhancements in the sulci of the left frontal, parietal, and temporal lobes (D). E, F: MRI 20 days after surgery. MRI/FLAIR shows the disappearance of hyperintensity in the sulci (E), while MRI/Gd VWI still demonstrates a linear enhancement in the left temporal lobe (F). G, H: MRI 3 months after surgery. Both MRI/FLAIR (G) and MRI/Gd VWI (H) indicate the disappearance of the postoperative changes. The white arrow indicates sulci hyperintensity or sulci enhancement.

peared in the left frontal lobe on MRI/FLAIR 3 days after surgery (Fig. 5C), sulci enhancement occurred on MRI/Gd VWI in the left frontal, parietal, and temporal lobes (Fig. 5D). The maximum scores of both sulci hyperintensity and sulci enhancement were observed 3 days after surgery. On MRI/FLARI 20 days after surgery, sulci hyperintensity disappeared (Fig. 5E), but MRI/Gd VWI still showed sulci enhancement in the temporal lobe (Fig. 5F). Three months after surgery, both sulci hyperintensity (Fig. 5G) and sulci enhancement disappeared completely (Fig. 5H).

DISCUSSION

This postoperative MRI study in patients with moyamoya disease showed that the sulci enhancement score on MRI/Gd VWI correlated well with the sulci hyperintensity score on MRI/FLAIR and that the peak of the former preceded the peak of the latter. Cortical hyperintensity on MRI/FLAIR was associated with surface enhancement above the same area on MRI/Gd VWI. To the best of our knowledge, this is the first study to report postoperative changes on MRI/Gd VWI in patients with moyamoya disease after bypass surgery.

Changes on MRI/FLAIR after surgery in moyamoya disease

In the present study, sulci hyperintensity on MRI/FLAIR was observed in all 25 hemispheres (100%) subjected to bypass surgery, and cortical hyperintensity was observed in 7 of the 25 hemispheres (28%). In patients with moyamoya disease, the sulci hyperintensity on MRI/FLAIR is known as the ivy sign, which reflects decreased CBF [1, 9]. The appearance of sulci hyperintensity on MRI/FLAIR has been reported after surgery for moyamoya disease. While the mechanisms of postoperative sulci hyperintensity have not been elucidated, postoperative sulci hyperintensity has been reported to be associated with hyperperfusion syndrome [2].

As postoperative cortical hyperintensity has been reported as a cortical hyperintensity belt sign [10, 11], the cortical hyperintensity belt sign in those reports represented hyperintensity in both the cortex and the sulci, which were described separately in this study. The postoperative sulci hyperintensity possibly reflects substantial changes in blood vessels, the arachnoid membrane, subarachnoid space, pia mater, and/or subpial space in the sulci; then, its nature might differ from that of cortical hyperintensity. Therefore, in the present study, we differentiated between sulci and cortical hyperintensity.

Changes on MRI/Gd VWI after surgery in moyamoya disease

The present study revealed sulci enhancement on MRI/Gd VWI after bypass surgery and that the peak of sulci enhancement preceded the peak of sulci hyperintensity on MRI/FLAIR. Several studies have reported MRI/Gd VWI findings in patients treated with conservative therapy and in the preoperative period and/or in the postoperative period over the long-term in patients treated with surgery [12, 13]. MRI/Gd T1-WI was more sensitive than MRI/FLAIR in

showing sulci intensity changes in children with moyamoya disease [14]. The present study is the first report to reveal postoperative changes on MRI/Gd VWI in patients who underwent bypass surgery for moyamoya disease. Our study demonstrated sulci enhancement on MRI/Gd VWI, which is related to sulci hyperintensity on MRI/FLAIR, and its peak was observed before the peak of the sulci hyperintensity. This study also demonstrated cortical surface enhancement on MRI/Gd VWI associated with cortical hyperintensity, which was observed just beneath the surface enhancement, on MRI/FLAIR. The sulci enhancement was seen along the arteries in the sulci, which suggests that the enhancement was located in the arterial wall [15]. In normal subjects, arterial wall enhancement is not observed on MRI/Gd VWI. Arterial wall enhancement on MRI/Gd VWI has been reported in cases of cerebral vasculitis and acute stage thrombosis [16, 17]. A suspected mechanism of arterial wall enhancement is a blood-brain barrier (BBB) impairment in the arterial wall [18]. The postoperative sulci hyperintensity on MRI/FLAIR could be related to the sulci enhancement on MRI/Gd VWI, which possibly reflects the BBB impairment in the arterial wall within the sulci. Narducci A. *et al.* reported extravasation of fluorescein onto the brain surface during the late phase in intraoperative video angiography during bypass surgery in patients with moyamoya disease [19]. They speculated that BBB disruption occurs in patients with moyamoya disease.

Several studies have suggested the occurrence of a BBB impairment in patients with moyamoya disease. Mutations in ring finger protein 213 (*RNF213* gene located on chromosome 17q25), are known to be responsible for moyamoya disease [20]. Roy *et al.* reported a study using *RNF213*-deficient human-derived brain endothelial cells, which demonstrated that *RNF213* mutations increased BBB permeability [21]. They also showed that loss of the *RNF213* gene decreased expression of the platelet endothelial cell adhesion molecule-1 and claudin 5 genes and increased the secretion of pro-inflammatory cytokines, such as interleukin (IL)-6 and IL-8, which may be related to BBB dysfunction. A significant increase in matrix metalloproteinase (MMP)-9 in the plasma of patients with moyamoya disease has been reported [22, 23]. MMPs, including MMP-9, which degrade both extracellular matrix proteins and tight junctions, also cause BBB dysfunction [24]. These reports suggested that patients with moyamoya disease are prone to BBB dysfunction. A suspected mechanism of postoperative hyperperfusion after bypass surgery in moyamoya disease is impairment of blood flow autoregulation, which is caused by chronic blood flow reduction [25]. A possible mechanism of the arterial wall enhancement in the sulci demonstrated in this study is as follows: the sudden increases in blood flow into the brain caused by bypass surgery along with dysfunction of blood flow autoregulation in a patient with moyamoya disease, in which the BBB is already impaired, causes leakage of contrast medium into the arterial wall.

The present study demonstrated that cortical hyperintensity on MRI/FLAIR is related to surface enhancement on MRI/Gd VWI. In cases of posterior reversible encephalopathy syndrome (PRES), the cortex shows hyperintensity on MRI/FLAIR, which is associated with nearby cortical surface enhancement on MRI/Gd T1-WI with enhancement in the sulci [26], which is similar to the cortical hyperintensity in the present study of postoperative moyamoya disease. The relationship between cortical surface enhancement on MRI/Gd T1-WI and cortical hyperintensity on MRI/FLAIR in the studies of PRES might be similar to the relationship between surface enhancement on MRI/Gd VWI and cortical hyperintensity on MRI/FLAIR in the present study. A reported mechanism of cortical hyperintensity observed in PRES is vasogenic edema, which might be related to the cortical hyperintensity in this study.

The more frequent occurrence of postoperative hyperperfusion syndrome was observed in patients with hemispheres showing the higher sulci enhancement scores significantly. In the future, this finding might apply to postoperative management to prevent hyperperfusion syndrome, such as strict blood pressure control in patients showing strong sulci enhancement.

Study limitations

The small number of patients in this retrospective study conducted at a single institution might have resulted in bias and incorrect results, especially regarding the cortical hyperintensity on MRI/FLAIR because of the small number of patients who showed cortical hyperintensity. Slow flow in arteries sometimes mimics arterial wall enhancement seen on MRI/Gd VWI. Sulci enhancement occurred in the areas supplied by the bypass surgery, in which blood flow was supposed to increase instead of decrease. In the hemisphere contralateral to the operated hemisphere, sulci enhancement was never observed in this series. The postoperative sulci enhancement disappeared over the long-term after surgery. Therefore, sulci enhancement on MRI/Gd VWI probably represents arterial wall enhancement but not arterial lumen enhancement caused by slow flow.

CONCLUSIONS

On MRI/Gd VWI, the observance of sulci enhancement related to sulci hyperintensity on MRI/FLAIR in the early phase after bypass surgery in patients with moyamoya disease, and the peak of the former preceded the peak of the latter. Surface enhancement on MRI/Gd VWI was related to the occurrence of cortical hyperintensity on MRI/FLAIR. These findings probably reflect increased BBB vulnerability after bypass surgery in moyamoya disease.

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STATEMENT OF CONFLICT

None.

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REFERENCES

1) Mori N, Mugikura S, Higano S, *et al.* The leptomeningeal "ivy sign" on fluid-attenuated inversion recovery MR imaging in

Moyamoya disease: a sign of decreased cerebral vascular reserve?. *AJNR Am J Neuroradiol*. 2009; 30(5): 930–935.
2) Horie N, Morikawa M, Morofuji Y, *et al*. De novo ivy sign indicates postoperative hyperperfusion in moyamoya disease. *Stroke*. 2014; 45(5): 1488–1491.
3) Roder C, Hauser TK, Ernemann U, Tatagiba M, Khan N, Bender B. Arterial wall contrast enhancement in progressive moyamoya disease. *J Neurosurg*. 2019; 132(6): 1845–1853.
4) Research Committee on the Pathology and Treatment of Spontaneous Occlusion of the Circle of Willis; Health Labour Sciences Research Grant for Research on Measures for Infractable Diseases. Guidelines for diagnosis and treatment of moyamoya disease (spontaneous occlusion of the circle of Willis). *Neurol Med Chir (Tokyo)*. 2012; 52(5): 245–266.
5) Zhao WG, Luo Q, Jia JB, Yu JL. Cerebral hyperperfusion syndrome after revascularization surgery in patients with moyamoya disease. *Br J Neurosurg*. 2013; 27(3): 321–325.
6) Barber PA, Demchuk AM, Zhang J, Buchan AM. Validity and reliability of a quantitative computed tomography score in predicting outcome of hyperacute stroke before thrombolytic therapy. ASPECTS Study Group. Alberta Stroke Programme Early CT Score [published correction appears in Lancet 2000 Jun 17; 355(9221): 2170]. *Lancet*. 2000; 355(9216): 1670–1674.
7) Wang LX, Wang H, Hao FB, *et al*. Ivy Sign in Moyamoya Disease: A Comparative Study of the FLAIR Vascular Hyperintensity Sign Against Contrast-Enhanced MRI. *AJNR Am J Neuroradiol*. 2021; 42(4): 694–700.
8) Muraoka S, Taoka T, Kawai H, *et al*. Changes in Vessel Wall Enhancement Related to the Recent Neurological Symptoms in Patients with Moyamoya Disease. *Neurol Med Chir (Tokyo)*. 2021; 61(9): 515–520.
9) Kaku Y, Iihara K, Nakajima N, *et al*. The leptomeningeal ivy sign on fluid-attenuated inversion recovery images in moyamoya disease: positron emission tomography study. *Cerebrovasc Dis*. 2013; 36(1): 19–25.
10) Hamano E, Kataoka H, Morita N, *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): 1–7.
11) Takemoto Y, Kawano T, Ohmori Y, *et al*. Hemodynamic study about cortical hyperintensity belt sign after direct bypass surgery for moyamoya disease. *J Clin Neurosci*. 2020; 74: 124–129.
12) Muraoka S, Araki Y, Taoka T, *et al*. Prediction of Intracranial Arterial Stenosis Progression in Patients with Moyamoya Vasculopathy: Contrast-Enhanced High-Resolution Magnetic Resonance Vessel Wall Imaging. *World Neurosurg*. 2018; 116: e1114–e1121.
13) Kathuveetil A, Sylaja PN, Senthilvelan S, Kesavadas C, Banerjee M, Jayanand Sudhir B. Vessel Wall Thickening and Enhancement in High-Resolution Intracranial Vessel Wall Imaging: A Predictor of Future Ischemic Events in Moyamoya Disease. *AJNR Am J Neuroradiol*. 2020; 41(1): 100–105.
14) Yoon HK, Shin HJ, Chang YW. "Ivy sign" in childhood moyamoya disease: depiction on FLAIR and contrast-enhanced T1-weighted MR images. *Radiology*. 2002; 223(2): 384–389.
15) Jung SC, Kang DW, Turan TN. Vessel and Vessel Wall Imaging. *Front Neurol Neurosci*. 2016; 40: 109–123.
16) Mandell DM, Mossa-Basha M, Qiao Y, *et al*. Intracranial Vessel Wall MRI: Principles and Expert Consensus Recommendations of the American Society of Neuroradiology. *AJNR Am J Neuroradiol*. 2017; 38(2): 218–229.
17) Arnett N, Pavlou A, Burke MP, Cucchiara BL, Rhee RL, Song JW. Vessel wall MR imaging of central nervous system vasculitis: a systematic review. *Neuroradiology*. 2022; 64(1): 43–58.
18) Renú A, Laredo C, Lopez-Rueda A, *et al*. Vessel Wall Enhancement and Blood-Cerebrospinal Fluid Barrier Disruption After Mechanical Thrombectomy in Acute Ischemic Stroke. *Stroke*. 2017; 48(3): 651–657.
19) Narducci A, Yasuyuki K, Onken J, Blecharz K, Vajkoczy P. In vivo demonstration of blood-brain barrier impairment in Moyamoya disease. *Acta Neurochir (Wien)*. 2019; 161(2): 371–378.
20) Kamada F, Aoki Y, Narisawa A, *et al*. A genome-wide association study identifies RNF213 as the first Moyamoya disease gene. *J Hum Genet*. 2011; 56(1): 34–40.
21) Roy V, Ross JP, Pépin R, *et al*. Moyamoya Disease Susceptibility Gene *RNF213* Regulates Endothelial Barrier Function. *Stroke*. 2022; 53(4): 1263–1275.
22) 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(5): 476–480.
23) Ishii D, Matsushige T, Okazaki T, *et al*. Marked Changes in Blood-Brain Barrier Biomarkers After Direct Bypass Surgery for Moyamoya Angiopathy: Preliminary Study. *World Neurosurg*. 2018; 120: e611–e616.
24) Rempe RG, Hartz AMS, Bauer B. Matrix metalloproteinases in the brain and blood-brain barrier: Versatile breakers and makers. *J Cereb Blood Flow Metab*. 2016; 36(9): 1481–1507.
25) Zhao WG, Luo Q, Jia JB, Yu JL. Cerebral hyperperfusion syndrome after revascularization surgery in patients with moyamoya disease. *Br J Neurosurg*. 2013; 27(3): 321–325.
26) Kastrup O, Schlamann M, Moenninghoff C, Forsting M, Goericke S. Posterior Reversible Encephalopathy Syndrome: The Spectrum of MR Imaging Patterns. *Clin Neuroradiol*. 2015; 25(2): 161–171.