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Original Article

Reversible sulcal fluid-attenuated inversion recovery hyperintensity after combined bypass surgery for moyamoya disease – A "crevasse" sign

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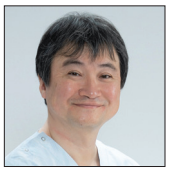

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ABSTRACT

Background: Transient fluid-attenuated inversion recovery (FLAIR) hyperintensity is often observed on the operated brain surface after direct or combined bypass surgery for moyamoya disease, but its pathophysiology and clinical significance are still obscure. This study was aimed to clarify the underlying mechanism and clinical significance.

Methods: This prospective study included 106 hemispheres of 61 patients with moyamoya disease and analyzed their radiological findings before and after combined bypass surgery. This study also included 11 patients who underwent superficial temporal artery to middle cerebral artery anastomosis for occlusive carotid artery diseases as the controls. Magnetic resonance imaging examination was serially repeated, and cerebral blood flow was measured before and after surgery. Signal intensity ratio (SIR) in the cortical sulci and cortex to the adjacent white matter on FLAIR images was calculated, and the postoperative SIR changes were semi-quantitatively evaluated to assess the temporal profile of postoperative FLAIR hyperintensity.

Results: Postoperative FLAIR hyperintensity occurred within the cortical sulci on the operated hemispheres in all moyamoya patients but not in patients with occlusive carotid artery diseases. SIR values started to increase immediately after surgery, peaked at about 4-fold at 4–13 days post-surgery, then declined, and recovered to baseline values over 28 days or later. The magnitude of this phenomenon was proportional to the severity of cerebral ischemia but not to postoperative hyperperfusion.

Conclusion: Reversible sulcal FLAIR hyperintensity specifically occurs in the operated hemispheres after direct bypass surgery for moyamoya disease. This "*crevasse sign*" may represent the mixture of the extensive leakage of oxygen and proteins from the pial arteries into the CSF.

Keywords: Bypass surgery, Cerebrospinal fluid, Cortical sulcus, Fluid-attenuated inversion recovery, Moyamoya disease, Oxygen

INTRODUCTION

Moyamoya disease is a unique cerebrovascular disease characterized by a progressive occlusion of the terminal portion of the internal carotid artery and its main branches. Moyamoya disease occurs in both children and adults and provokes a variety of cerebrovascular events, including transient ischemic attack, ischemic stroke, and hemorrhagic stroke.[8,23] Surgical revascularization

is widely accepted to improve cerebral hemodynamics and reduce the risk of further cerebrovascular events. Surgical revascularization includes direct, indirect bypass, and combined bypass. Of these, both direct and combined bypass can immediately improve cerebral hemodynamics by providing bypass flow through the superficial temporal artery to middle cerebral artery (STA-MCA) anastomosis.[8] These drastic hemodynamic changes may have various impacts on the operated brain, such as postoperative hyperperfusion.[25] According to recent reports, the signal intensity on fluid-attenuated inversion recovery (FLAIR) images is known to transiently elevate on the operated brain surface after STA-MCA anastomosis for moyamoya disease. Horie *et al*. first pointed out this unique phenomenon and speculated it as the dilated pial vessels in the brain surface. They concluded that it was closely related to postoperative hyperperfusion.[6] On the other hand, Hamano *et al*. hypothesized that this phenomenon represented cortical vasogenic edema and was associated with transient neurological deficits.[5] However, the pathophysiology and clinical significance are still to be debated. This is partly because previous studies have been conducted with only visual inspection.

Based on these observations, this prospective study aimed to clarify the underlying mechanism and clinical significance of transient FLAIR hyperintensity after combined bypass surgery for moyamoya disease. For this purpose, we precisely specified the location of this phenomenon and semi-quantitatively evaluated the temporal and spatial profiles of this phenomenon. Furthermore, we assessed the relationship between this phenomenon and cerebral hemodynamics before and after surgery. The results would be valuable to understanding the underlying pathophysiology of this unique phenomenon in moyamoya disease to improve our perioperative management.

MATERIALS AND METHODS

Patients

This prospective study was approved by the Institutional Review Board of our hospital and was conducted according to the Declaration of Helsinki II. This study prospectively enrolled 61 patients with moyamoya disease and analyzed their radiological findings before and after surgical revascularization. All of them were diagnosed with moyamoya disease according to the diagnostic criteria set by the Research Committee on moyamoya Disease of Japan[22] and underwent STA-MCA anastomosis and indirect bypass, encephalo-duro-myo-arterio-pericranial synangiosis (EDMAPS) at our hospital.[9,12]

This study included 61 patients with moyamoya disease. There were 22 males and 39 females, including 21 children (6–18 years) and 40 adults. Mean age was 8.7 ± 4.3 years in children and 38.6 ± 8.7 years in adults. Surgical revascularization was performed on 106 hemispheres, including 37 hemispheres in children and 69 in adults. As the controls, this study also included 11 patients who underwent standard STA-MCA anastomosis for occlusive carotid artery disease. All of them were male, and their mean age was 71.1 ± 8.8 years.

Magnetic resonance imaging (MRI)

Using a 1.5-Tesla scanner (Magnetom Avanto, Siemens, Erlangen, Germany), all MRIs were obtained 3 days before surgery and were repeated 7 times after surgery. The timing of MR imaging was 0–3, 4–8, 9–13, 14–18, 18–22, 23–27, and 28 days after surgery. In addition to standard T1-, T2-, and diffusion-weighted images, the FLAIR image was acquired with the following parameters: TR/effective TE, 9,000/109; inversion time, 2500 ms; voxel size = 1.0 × 0.9 × 6.0 mm; FOV = 220 mm; parallel imaging mode = GRAPPA; accelerating factor = 2; and section thickness, 6 mm. Susceptibility-weighted image (SWI) was also acquired with the following parameters: TR/TE = 48/40 ms, FA = 15°; voxel size = 0.9 × 0.9 × 1.8 mm; FOV = 230 mm, parallel imaging mode = GRAPPA; accelerating factor = 3; and section thickness, 1.8 mm. Two certified neurosurgeons (DK and SK) who were blinded to clinical data independently evaluated the data and resolved disagreements by consensus.

SPECT

Before surgery, cerebral blood flow (CBF) before and after intravenous injection of 10-mg/kg acetazolamide (ACZ) was quantitatively measured with the ^{123}I- *N*-isopropyl-p-iodoamphetamine injection and single-scan autoradiographic technique (GCA-9300/DI; Toshiba).[13,14] To evaluate cerebral hemodynamics, the regions of interest (ROIs) were symmetrically placed in the ipsilateral and contralateral middle cerebral artery (MCA) territories. As described previously, cerebrovascular reactivity (CVR) to ACZ was quantitatively calculated as:

$CVR\ (\%) = 100 \times (CBF_{ACZ} - CBF_{rest})/CBF_{rest}$, where CBF_{rest} and CBF_{ACZ} represent CBF before and after intravenous injection of ACZ, respectively. CBF was judged as reduced when the value was lower than 27 ml/min/100g, and CVR was judged as reduced when the value was lower than <14%. According to Kuroda's classification, the hemispheres with normal CBF and reduced CVR were categorized into Type 2 ischemia, and those with reduced CBF and CVR were categorized into Type 3 ischemia.[10,11]

CBF measurement was repeated just after surgery and 2- and 7-days post-surgery. CBF was rated as abnormally

elevated when the CBF in the operated MCA territory was higher than 150% of blood flow in the ipsilateral cerebellum.[25]

Bypass surgery

STA-MCA anastomosis and EDMAPS were performed on the hemispheres with the hemispheres with normal CBF but reduced CVR (Kuroda's Type 2, $n = 14$) and those with reduced CBF and CVR (Kuroda's Type 3, $n = 92$).[9,12] Briefly, the frontal and parietal branches of the STA were harvested from the scalp. Following frontotemporal craniotomy extending to the medial frontal area, the dura mater was widely opened to expose the brain surface. One or two cortical branches of the MCA feeding the frontal and temporal branches were selected as the recipients. Using 10-0 or 11-0 nylon threads, the distal end of STA was anastomosed to the cortical branches of the MCA in an end-to-side fashion. Clamping time ranged from 15 min to 30 min. The patency of the bypass graft was confirmed with indocyanine green video angiography during surgery. Finally, the dural window was covered with the vascularized dura mater, temporal muscle, and frontal pericranium as the indirect bypass procedure.[9,12]

The patients with atherosclerotic carotid artery diseases underwent STA-MCA single or double anastomosis onto the hemispheres with Kuroda's Type 3 ischemia ($n = 11$).[10,11]

Semi-quantitative analysis of transient FLAIR hyperintensity

The temporal profile in the FLAIR intensity was semi-quantitatively analyzed using each patient's set of FLAIR images. For this purpose, the 2.5-mm-diameter circular ROIs were placed on the cortex, adjacent white matter, and cortical sulci of each operated hemisphere at levels of the basal ganglia and the body of the lateral ventricle [Figure 1]. The average crude signal intensity in the cortex and cerebral sulci was normalized by calculating the signal intensity ratio (SIR) to that in the adjacent white matter according to the following formula:

$$SIR_{Cortex} = Signal\ intensity_{\ cerebral\ cortex}/Signal\ intensity_{\ white\ matter}$$

$$SIR_{Sulci} = Signal\ intensity_{\ cortical\ sulci}/Signal\ intensity_{\ white\ matter}$$

Then, the postoperative changes in SIR (ΔSIR) were calculated according to the following formula:

$$\Delta\ SIR_{Cortex} = (SIR_{Cortex\ postop.} - SIR_{Cortex\ preop.})/SIR_{Cortex\ preop.}$$

$$\Delta\ SIR_{Sulci} = (SIR_{Sulci\ postop.} - SIR_{Sulci\ preop.})/SIR_{Sulci\ preop.}$$

In these formulas, the white matter was selected as the internal reference because it is known that oxygen (O_2) does not have no paramagnetic effects on the white matter.[2]

Statistical analysis

Continuous variables were expressed as mean ± standard deviation. Significant differences were set at $P < 0.05$. Variables were compared between the two groups using Mann–Whitney U-test.

RESULTS

Location and temporal profile

As shown in Figure 1, postoperative FLAIR hyperintensity emerged to varying degrees on the operated hemispheres in all moyamoya patients but not in patients with occlusive carotid artery diseases. The appearance of postoperative FLAIR hyperintensity was completely different from the "ivy" sign that is often observed as the result of slow flow in the arteries. None of the moyamoya patients presented any signs of meningitis after surgery.

To specify the location of postoperative FLAIR hyperintensity in moyamoya disease, magnified MRI images of the brain surface were evaluated. Preoperative FLAIR images showed no abnormality on the brain surface [Figure 2a], but postoperative FLAIR images demonstrated diffuse hyperintensity in the brain surface. Magnified images clearly showed that the FLAIR hyperintensity occurred within the cortical sulci but not in the cortex or cortical vessels of the brain surface [Figure 2b]. On simultaneous T2*-weighted images, the FLAIR hyperintensity was not observed as a low-signal intensity, suggesting that the FLAIR hyperintensity did not represent postoperative clot accumulation within the cortical sulci [Figure 2c].

To confirm these results of visual inspection, the signal changes in the cortical sulci and cortex were semi-quantitatively analyzed. Figure 2d demonstrates the temporal profile of ΔSIR_{Sulci} and ΔSIR_{Cortex} values in the ipsilateral MCA territory after bypass surgery for moyamoya disease. The ΔSIR_{Sulci} value started to increase immediately after surgery, peaked at about 4-fold 4–13 days post-surgery, then declined and recovered to baseline values over 28 days or later. On the other hand, ΔSIR_{Cortex} value showed no significant changes during 28 days after surgery. Therefore, it is most likely that postoperative FLAIR hyperintensity results from an increase in the intensity of cerebrospinal fluid (CSF) within the cortical sulci.

Impacts of cerebral hemodynamics

To investigate the impact of the severity of cerebral ischemia on postoperative FLAIR hyperintensity, the ΔSIR_{Sulci} value was compared between Type 2 and Type 3 hemispheres. As shown in Figure 3, the temporal profile of ΔSIR_{Sulci} value was similar between Type 2 and Type 3 hemispheres, but the ΔSIR_{Sulci} values were significantly higher in the Type 3 hemispheres than in the

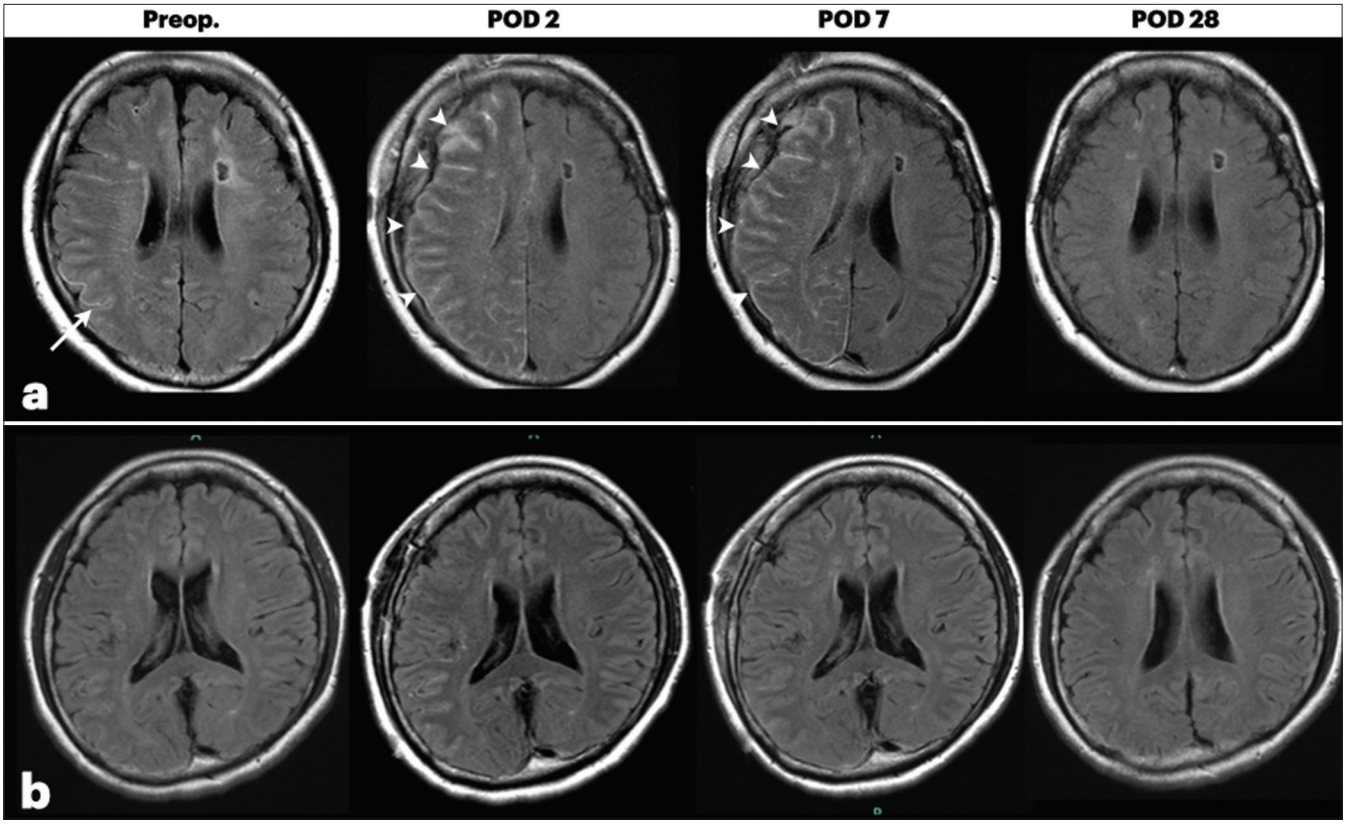

Figure 1: (a) Representative series of FLAIR images before and after bypass surgery on the right side for a 41-year-old female with moyamoya disease. In this case, typical "ivy" sign could be observed on the surface of parietal lobe (arrow). However, the "ivy" sign completely disappeared and postoperative FLAIR intensity was observed widely on the right hemispheres at 2 and 4 days after STA-MCA anastomosis and EDMAPS onto the right side (arrowheads). Postoperative FLAIR hyperintensity completely disappeared 28 days post-surgery. (b) Representative series of FLAIR images before and after bypass surgery on the right side for a 72-year-old male with internal carotid artery occlusion. Note that these findings were not found on the operated hemisphere after right STA-MCA anastomosis. FLAIR: Fluid attenuated inversion recovery, STA-MCA: Superficial temporal artery to middle cerebral artery, EDMAPS: Encephalo-duro-myo-arterio-pericranial synangiosis.

Type 2 hemispheres through 28 days after surgery ($P < 0.01$ at each time point). For example, the ΔSIR_{Sulci} value at 4–8 days post-surgery was 4.2 ± 0.4 and 2.4 ± 0.3 in hemispheres with Type 2 and Type 3 hemispheres, respectively ($P < 0.01$).

In a certain subgroup of moyamoya patients, postoperative FLAIR hyperintensity was observed in the ipsilateral occipital lobe outside the craniotomy. The ipsilateral posterior cerebral artery (PCA) was involved in a majority of them [Figure 4]. Therefore, we compared the temporal profile of ΔSIR_{Sulci} value in the occipital lobe between moyamoya patients with PCA stenosis and those without. As a result, ΔSIR_{Sulci} value significantly increased through 28 days after surgery in the PCA-involved group but not in the non-PCA-involved group ($P < 0.01$ at each time point; Figure 4e).

Postoperative hyperperfusion

Finally, we evaluated the impact of postoperative hyperperfusion on postoperative FLAIR hyperintensity in moyamoya disease. Postoperative hyperperfusion occurred in 37 (40.2%) of 92 Type 3 hemispheres. We compared ΔSIR_{Sulci} values between the hemispheres with postoperative hyperperfusion ($n = 37$) and those without ($n = 55$). The temporal profile was very similar between the two groups, and there were no significant differences in the ΔSIR_{Sulci} value at each time point. The peak $\Delta\ SIR_{Sulci}$ value at 4–8 days post-surgery was 4.2 ± 0.4 and 4.0 ± 0.3 in the hemispheres with hyperperfusion and those without, respectively [Figure 5].

DISCUSSION

This study mainly explores the following observations: *First*, postoperative FLAIR hyperintensity represents the increase in signal intensity within the cerebral sulci but not in the cortex or blood vessels on the brain surface. The finding strongly suggests that this phenomenon represents a postoperative change in the environment of CSF space.

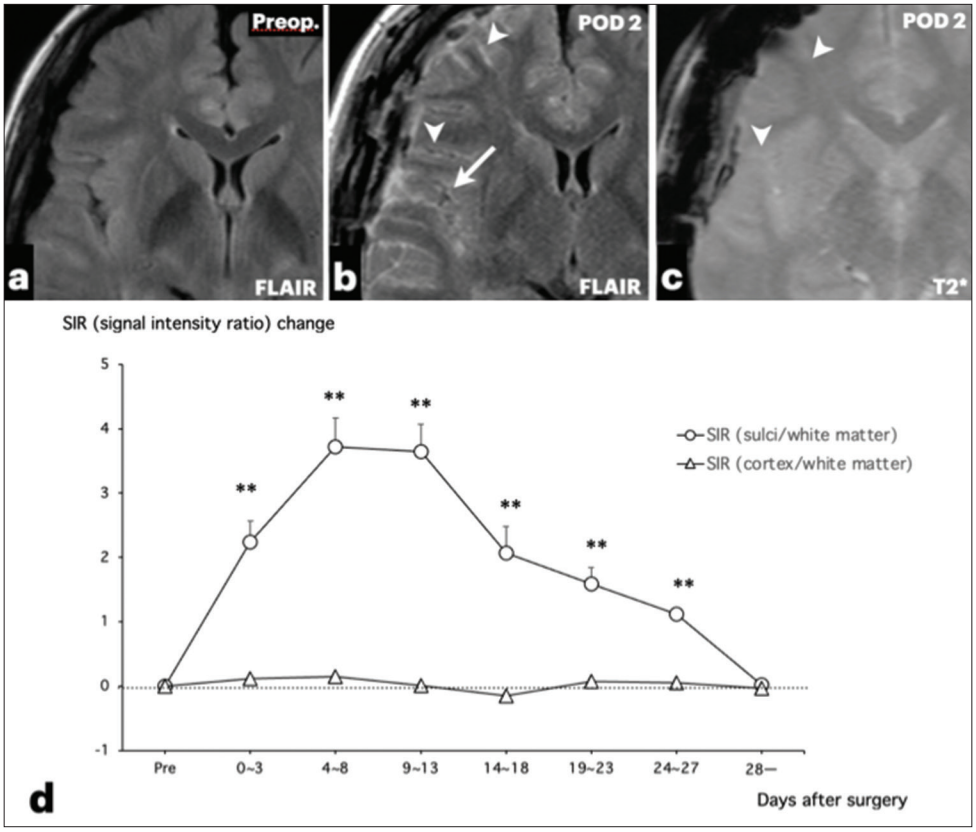

Figure 2: (a) Magnified FLAIR image before STA-MCA anastomosis and EDMAPS for a 35-year-old female with moyamoya disease. Note normal appearance before surgery. (b) Magnified FLAIR image at 2 days after surgery. Postoperative FLAIR hyperintensity was clearly observed within the cortical sulci at 2 days after STA-MCA anastomosis and EDMAPS (arrowheads). The cortical arteries within the cortical sulci are shown as low signal intensity because of flow void phenomenon on FLAIR image (arrow). (c) Magnified T2*-weighted MR at 2 days after surgery. The FLAIR hyperintensity was not detected as low signal intensity on T2*-weighted image, suggesting that this phenomenon is not due to postoperative blood accumulation within the cortical sulci (arrowheads). (d) A line graph shows the serial changes of signal intensity ratio (SIR) in the cortical sulci (open circle) and cortex (open triangle) in the MCA territory. In the cortical sulci, the SIR value started to increase within 3 days after STA-MCA anastomosis and EDMAPS, reaches the peak 4 to 13 days, and returns to the control level over 28 days post-surgery. However, the SIR values exhibited no significant changes throughout postoperative period in the cortex. **; $P<0.01$. FLAIR: Fluid attenuated inversion recovery, STA-MCA: Superficial temporal artery to middle cerebral artery, EDMAPS: Encephalo-duro-myo-arterio-pericranial synangiosis, SIR: Signal intensity ratio, POD: Postoperative day.

Second, the phenomenon is observed in moyamoya patients but not in patients with occlusive carotid artery diseases, indicating that this "sulcal" FLAIR hyperintensity occurs through the pathophysiology specific to moyamoya disease. *Third*, the degree of postoperative "sulcal" FLAIR hyperintensity depends on the severity of cerebral ischemia before surgery, which strongly suggests that preoperative cerebral ischemia may play a key role in the occurrence of the phenomenon. On the other hand, postoperative hyperperfusion is not related to the occurrence of postoperative FLAIR hyperintensity.

Sulcal FLAIR hyperintensity has been recognized to occur through the failure to suppress the CSF signal on FLAIR imaging and previously described as "hyperintense CSF," "leptomeningeal hyperintensity," or "hyperintensity within the subarachnoid space."[24] The finding has been reported in patients with abnormal CSF, such as subarachnoid hemorrhage and meningitis.[20,21] Considering the fact that the blood clots are detected as low signal on T2*-weighted images, however, postoperative blood accumulation in the cerebral sulci is unlikely to be the cause of sulcal FLAIR hyperintensity observed in this study because no

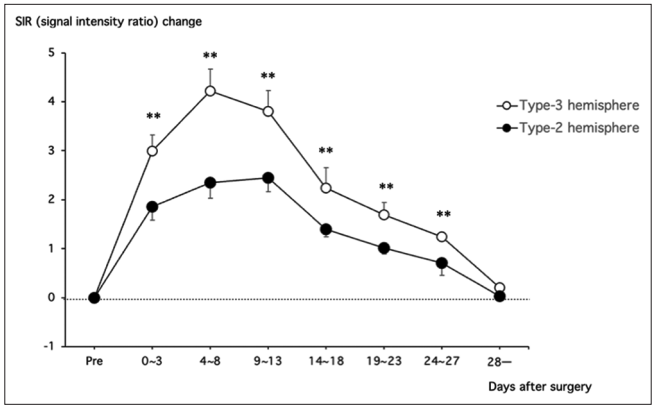

Figure 3: A line graph illustrates the serial changes of signal intensity ratio (SIR) in the cortical sulci after superficial temporal artery to middle cerebral artery anastomosis and encephalo-duro-myo-arterio-pericranial synangiosis for moyamoya hemispheres with Type 3 ischemia (*open circle*) and those with Type 2 ischemia (*closed circle*) in the middle cerebral artery territory. The SIR value was significantly higher in hemispheres with Type 3 ischemia than those with Type 2 ischemia within 28 days after surgery. $^{**}P < 0.01$.

abnormal low signal was not detected on T2*-weighted images. The possibility of meningitis can be excluded due to the lack of clinical evidence in the subjects. Indeed, sulcal FLAIR hyperintensity has been reported in patients without apparent CSF abnormalities under the following situations:

First, sulcal FLAIR hyperintensity may occur through the leakage of protein from blood to CSF after surgery. Thus, vascular permeability may be increased by the blood-brain barrier (BBB) in moyamoya disease. For example, gadolinium contrast is known to leak from the pial arteries into the CSF due to the BBB breakdown in the acute stage of ischemic stroke.[3,7] More interestingly, Narducci *et al*. observed the leakage of sodium fluorescein from the pial arteries to the CSF immediately after a direct bypass procedure for moyamoya patients.[19] These observations strongly suggest that direct bypass procedures may drastically increase the vascular permeability in the operated hemispheres and induce protein leakage into the CSF. FLAIR imaging is known to be very useful in detecting pathologic conditions that increase the protein concentrations in the CSF. Melhem *et al*. reported that protein concentration thresholds for CSF hyperintensity depend on the effective TE used in the FLAIR sequence. According to their results, this threshold is 250 mg/dL at 110 ms, which is very close to the 109 ms effective TE that we used in this study.[18] Considering the fact that normal protein concentrations are 10–40 mg/dL, it is unlikely that the protein concentration in CSF always rises above 250 mL/dL for more than 2 weeks after surgery, making it difficult to explain the cause of this sulcal FLAIR hyperintensity solely by the increase in protein concentration in the CSF.

Second, FLAIR imaging is also known to be sensitive to the changes in O_2 concentration in the CSF.[1,2,4] The normal O_2 tension in the CSF is known as 138 ± 46 mmHg under physiological conditions.[17] A 20-minute inhalation of 100% O_2 leads to a drastic increase of O_2 tension in the CSF from 124 ± 55 mmHg to 228 ± 18 mmHg.[26] When the fraction of inspiratory O_2 (FiO_2) is changed from 30% to 100%, CSF signal intensity significantly increases in all anesthetized patients.[4] Anzai also reported that a FiO_2 change from 20% to 100% induced a 4-–5.3-fold increase in CSF signal intensity in awake humans.[1] Very interestingly, this value is almost the same as the peak ΔSIR_{Sulci} value after bypass surgery for moyamoya disease in this study [Figures 2, 3, and 5]. Therefore, the direct bypass procedure for moyamoya disease may induce the partial pressure of O_2 in CSF ($P_{CSF}O_2$) through supplemental O_2 leakage from the pial arteries to the CSF and induce the elevation of CSF signal intensity on FLAIR images. This speculation is supported by the fact that the degree of postoperative FLAIR hyperintensity depends on the severity of cerebral ischemia before surgery. In moyamoya patients with PCA stenosis, furthermore, sulcal FLAIR hyperintensity also developed in the occipital lobe despite being outside the craniotomy area. To prove this hypothesis, we intraoperatively measured $P_{CSF}O_2$ before and after bypass surgery. $P_{CSF}O_2$ was significantly lower in moyamoya disease than in the controls. $P_{CSF}O_2$ was much lower in moyamoya disease than in occlusive carotid artery disease. Direct bypass procedures dramatically improved $P_{CSF}O_2$. Interestingly, post-bypass $P_{CSF}O_2$ changes were greater in moyamoya disease than in occlusive carotid artery disease.[15,16] This may explain why sulcal FLAIR hyperintensity emerges in moyamoya disease but not in occlusive carotid artery disease after surgery. Another reason may be that patients who undergo bypass surgery for occlusive carotid artery disease are often elderly and have significant brain atrophy, so the CSF within the cerebral sulci diffuses more easily than in moyamoya disease, which is more common in younger patients.

Based on these observations, we speculate that postoperative FLAIR hyperintensity may develop through the combination of a sudden elevation of $P_{CSF}O_2$ and a protein leakage into the CSF after direct bypass procedures. Such drastic changes may continue for about 2 weeks, followed by a gradual resolution. Finally, we would like to propose giving the name "*crevasse sign*" to this uncommon radiological phenomenon that is specifically manifested in moyamoya disease because this phenomenon can be observed in the subarachnoid space of the cerebral sulci and is morphologically very similar to a series of "crevasse" deeply carved into a glacier.

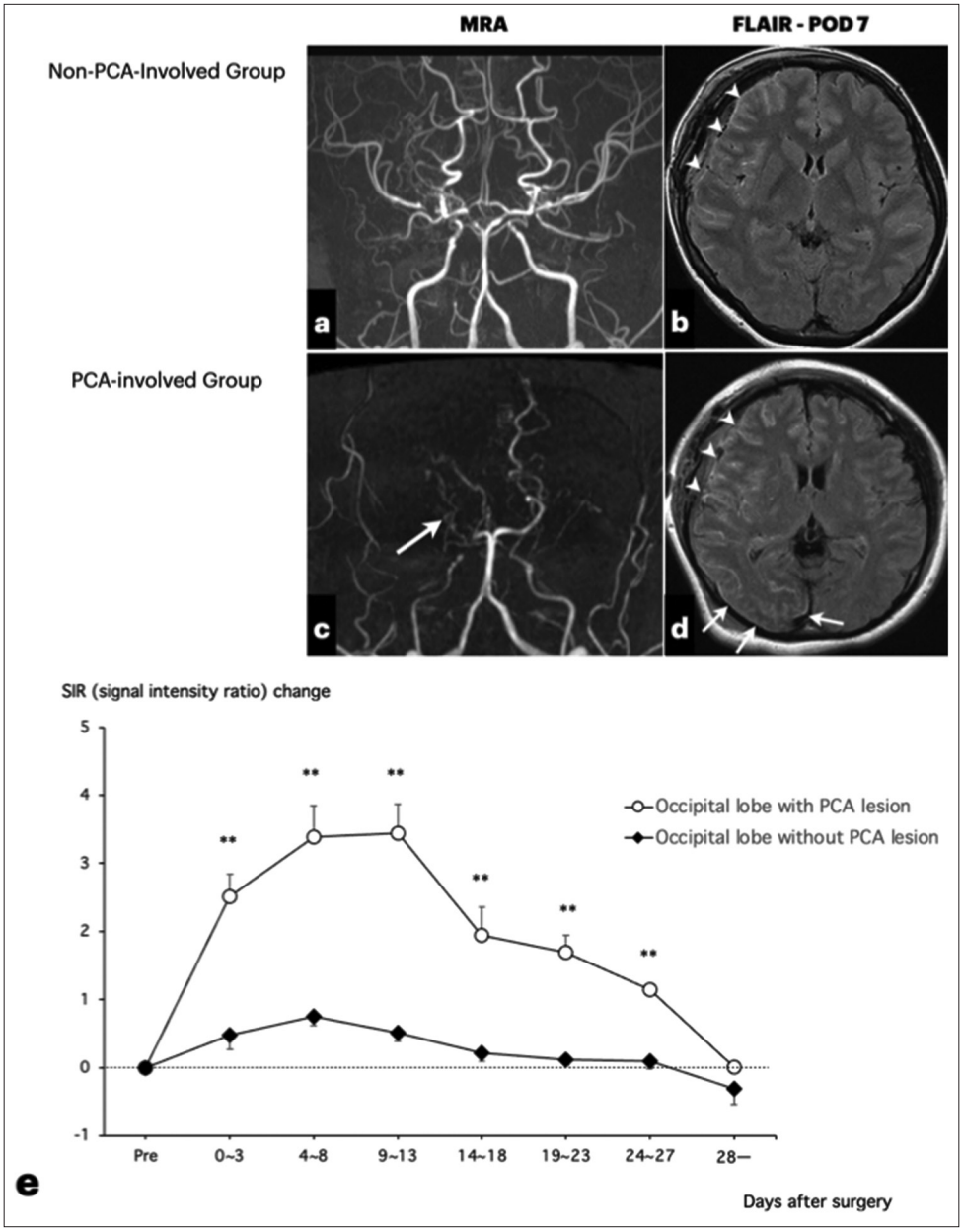

Figure 4: (a, b) Representative preoperative MR angiography and postoperative FLAIR images at 7 days after right STA-MCA anastomosis and EDMAPS for a 42-year-old moyamoya patient without PCA involvement. Note that postoperative FLAIR hyperintensity was limited in the MCA area (arrowheads). (c, d) Representative preoperative MR angiography and postoperative FLAIR images at 7 days after right STA-MCA anastomosis and EDMAPS for a 48-year-old moyamoya patient with PCA involvement. Note that the finding was observed in the occipital lobe (d, arrows) as well as in the MCA area (d, arrowheads) in patient with PCA involvement (c, arrow). (e) A line graph demonstrates the serial changes of signal intensity ratio (SIR) in the cortical sulci of the occipital lobe after STA-MCA anastomosis and EDMAPS for moyamoya patients with PCA involvement (open circle) and those without (closed diamond). Note that the SIR changes in the occipital lobe were significantly pronounced in patients with PCA involvement than in those without. **; $P<0.01$. FLAIR: Fluid attenuated inversion recovery, STA-MCA: Superficial temporal artery to middle cerebral artery, EDMAPS: Encephalo-duro-myo-arterio-pericranial synangiosis,SIR: Signal intensity ratio, PCA: Posterior cerebral artery, POD: Postoperative day.

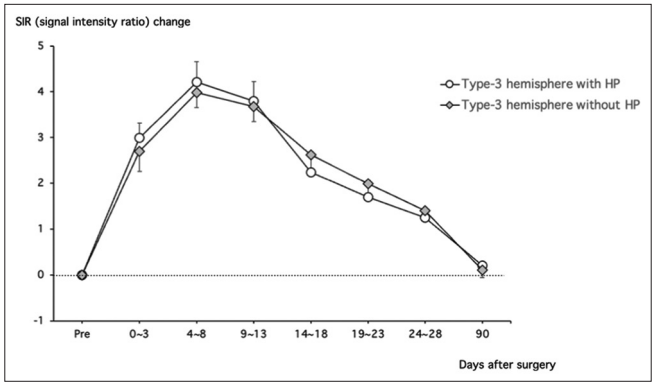

Figure 5: A line graph shows the serial changes of signal intensity ratio (SIR) in the cortical sulci after superficial temporal artery to middle cerebral artery anastomosis and encephalo-duro-myo-arterio-pericranial synangiosis in the middle cerebral artery area of moyamoya patients with Type 3 ischemia. Note that there were no significant differences in the SIR changes between the patients with postoperative hyperperfusion (*open circle*) and those without (*dashed diamond*) after surgery. $^{**}P < 0.01$.

CONCLUSION

Postoperative FLAIR hyperintensity specifically occurs in the subarachnoid space of the cerebral sulci on the operated hemispheres after direct bypass surgery for moyamoya disease. This "*crevasse sign*" may reflect an extensive leakage of O_2 and proteins from the pial arteries into the CSF through an increased vascular permeability in the areas exposed to preoperative cerebral ischemia.

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Ethical approval

The research/study was approved by the Institutional Review Board at Toyama University Hospital, number R2019057, dated August 30, 2019.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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