

Endoscopically Treated Subacute Subdural Hematoma Presenting Postoperative Cerebral Hyperperfusion Syndrome: Chronological Changes of Cerebral Blood Flow on Arterial Spin Labeling and Subcortical Low Intensity on Fluid-attenuated Inversion Recovery Images

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Abstract

Subacute subdural hematoma (SASDH) is a neurotraumatic entity. There are few reports of chronological changes of cerebral blood flow (CBF) on arterial spin labeling (ASL) and subcortical low intensity (SCLI) on fluid-attenuated inversion recovery (FLAIR) images of magnetic resonance imaging (MRI) observations from the injury onset, deterioration, to post-surgery. We reported a SASDH patient presenting postoperative cerebral hyperperfusion (CHP) syndrome with chronological changes of those findings. An 85-year-old woman fell and presented right ASDH. She was treated conservatively due to no neurological deficits. On day 3, ASL image revealed increased CBF against brain compression. On day 7, the CBF was normalized on ASL image, but SCLI was confirmed. On day 14, SCLI was strengthened. Then she developed left hemiparesis due to brain compression by SASDH. Considering age and comorbidities, we performed endoscopic hematoma removal under local anesthesia, and her neurological deficits improved after the surgery. On post-operative day 1, she newly presented left upper limb paresis. MRI revealed increased CBF and enhanced SCLI. We diagnosed CHP syndrome, and antihypertensive treatment improved the symptoms gradually. However, SCLI had been consistently observed, and CBF easily changed depending on the blood pressure, suggesting dysfunction of the CBF autoregulation. We showed the endoscopically treated SASDH patient with CBF's chronological changes on ASL images and SCLI on FLAIR images. Long-time brain compression would lead to dysfunction of the CBF autoregulation, and we should be careful about CHP syndrome after the endoscopic surgery for SASDH.

Keywords: arterial spin labeling, cerebral hyperperfusion, subcortical low intensity on fluid-attenuated inversion recovery, neuroendoscope, subacute subdural hematoma

Introduction

Subacute subdural hematoma (SASDH) has been reported to occur in approximately 10% of patients with acute SDH (ASDH) who are treated

conservatively.¹⁾ The outcomes are usually favorable after appropriate surgical treatment,¹⁻⁵⁾ but postoperative cerebral hyperperfusion syndrome (CHP) can rarely occur after surgical treatment for SASDH.⁶⁾ We treated a SASDH woman by endoscopic hematoma removal under local anesthesia, but she developed CHP syndrome postoperatively. We herein report her perioperative hemodynamic changes observed on the arterial spin labeling (ASL) images of the magnetic resonance imaging (MRI) from the injury to post-surgery. We also showed the perioperative

Received November 26, 2020; Accepted January 4, 2021

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subcortical low intensity (SCLI) changes on fluid-attenuated inversion recovery (FLAIR) images, which is possibly reflecting cytotoxic edema⁷⁾ and free radical production, associated with cerebral blood flow (CBF) increase and dysregulation.^{8–10)}

Case Report

An 85-year-old woman, who had hypertension and asthma, fell down, and had walking difficulty. Head computed tomography (CT) revealed right ASDH with 11 mm thickness (Fig. 1A). Her consciousness was well as Japan Coma Scale (JCS) 0 without any neurological deficits. Therefore, we treat her conservatively without blood pressure

reduction. On the next day, head CT revealed no hematoma expansion (Fig. 1B). She started rehabilitation and could walk with assistance. On day 3, T2-weighted image (T2WI) and FLAIR image did not show brain edema nor SCLI (blue arrowheads in Fig. 1C), and ASL image described increased CBF against the brain compression by the hematoma (circle in Fig. 1C). Her rehabilitation progressed favorably, and she could walk independently on day 6. T2WI and FLAIR image on day 7 also showed no brain edema, but mild SCLI was observed (yellow arrowheads in Fig. 1D). ASL image showed no left–right difference of the CBF (Fig. 1D). She was doing well, and we took MRI on day 14. FLAIR image revealed more apparent SCLI just under the

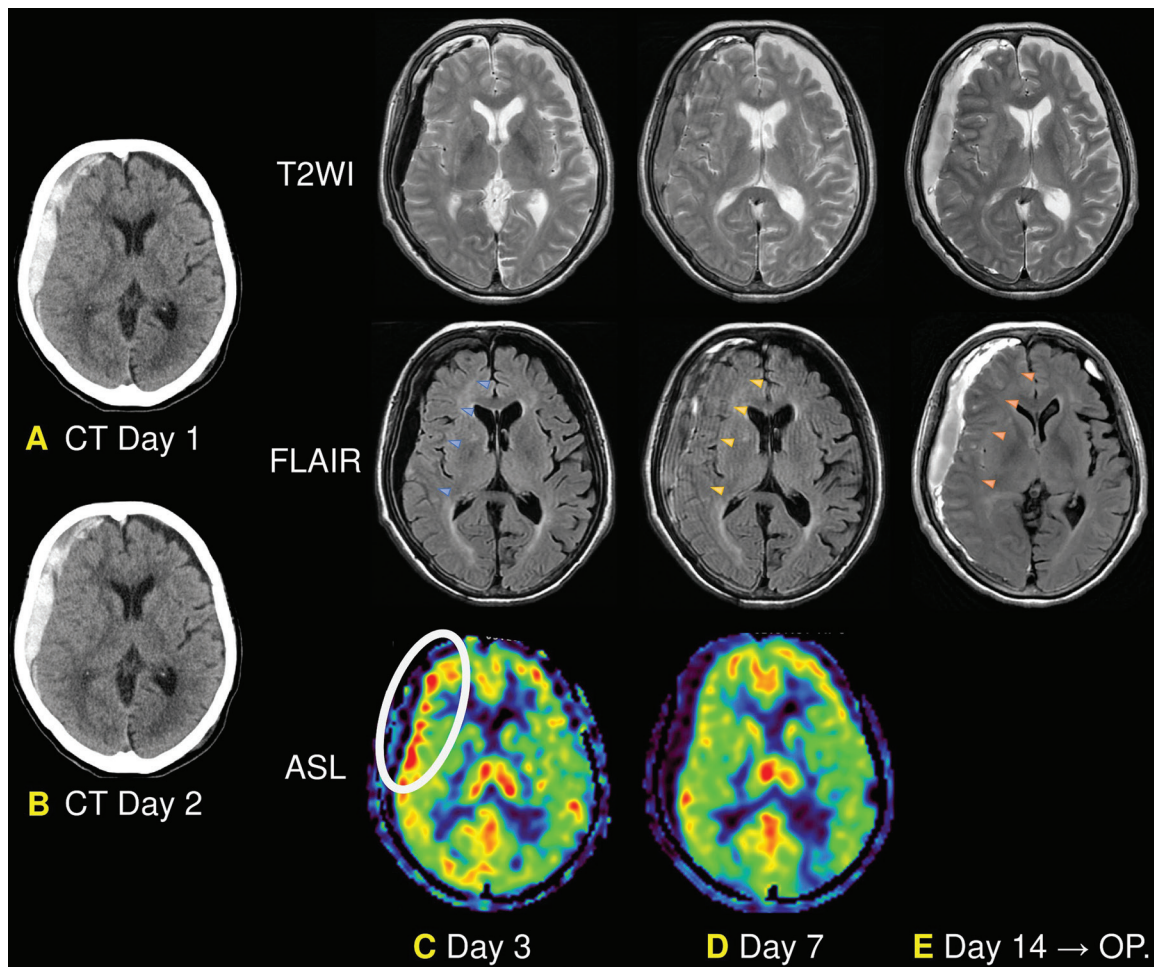


Fig. 1 Preoperative radiological images: Head CT on day 1 revealed right ASDH (A). On day 2, CT revealed no hematoma expansion (B). On day 3, T2WI and FLAIR image did not show brain edema nor SCLI (blue arrowheads in C), and ASL image described increased CBF against the brain compression (circle in C). On day 7, T2WI and FLAIR image showed no brain edema, but mild SCLI was observed (yellow arrowheads in D). ASL image showed no left–right difference of the CBF (E). On day 14, FLAIR image revealed more apparent SCLI just under the brain compression (red arrowheads in E), then we performed surgical hematoma removal. ASL: arterial spin labeling, CBF: cerebral blood flow, CT: computed tomography, FLAIR: fluid-attenuated inversion recovery, SCLI: subcortical low intensity, T2WI: T2-weighted image.

ASDH compressing the brain (red arrowheads in Fig. 1E). During MRI acquisition, she called us and appealed her paresis of left upper and lower extremities with the manual muscle test (MMT) 1/5. We took emergent CT (Fig. 2A) and determined to perform a surgical intervention.

Considering the age, comorbidities, and the urgency as well as the hematoma softness predicted by the high-intensity lesion in the T2WI (Fig. 1E), we determined to perform a burr hole and drainage surgery under local anesthesia. The patient's head was rotated contralateral to the hematoma side and placed on a horseshoe headrest. A 3-cm linear skin incision was made parallel to the coronal suture, and a burr hole was made on the point under which the hematoma thickness was largest (Fig. 2B). After a cruciate dural incision, we drained a small amount of the serous hematoma. However, most of the hematoma was too hard to be removed by drainage, and decompression seemed insufficient. Therefore, we enlarged the burr hole by the luer bone rongeurs, and introduced a rigid endoscope (A70960, 2.7 mm, 0° or 30° angle; Olympus Corporation, Tokyo, Japan) into the subdural space. Under the endoscopic observation, rigid hematoma (red arrow in Fig. 2C)

was evacuated by suction cannula (gray arrow in Fig. 2C). Some of the hematoma was too stiff to be evacuated (Fig. 2D), so we sometimes used micro-scissors to remove the hematoma as piecemeal (Fig. 2E). The hematoma just above the primary motor cortex seemed to be adequately removed, so we intentionally left some part of the hard hematoma in the parietal and posterior regions, which was not associated with her paresis (green arrow in Fig. 2F). As endoscopic findings, the outer membrane seemed red, suggesting inflammation¹¹⁾ (blue arrows in Fig. 2C, 2F). We closed the burr hole with a burr hole cap made of hydroxyapatite and sutured the skin. The operative time was 46 min, and the bleeding amount was 10 mL. The postoperative CT showed sufficient decompression (Fig. 2G) with small craniotomy (colored yellow in Fig. 2H). After the surgery, her paresis improved as MMT 4/5, and we maintained the blood pressure under 140 mmHg.

In the morning of day 15, her consciousness was well as JCS 0, and her paresis improved as MMT 5/5. We regulated her blood pressure under 180 mmHg after checking the CT. However, in the evening, she complained of paresis at her left upper limb with

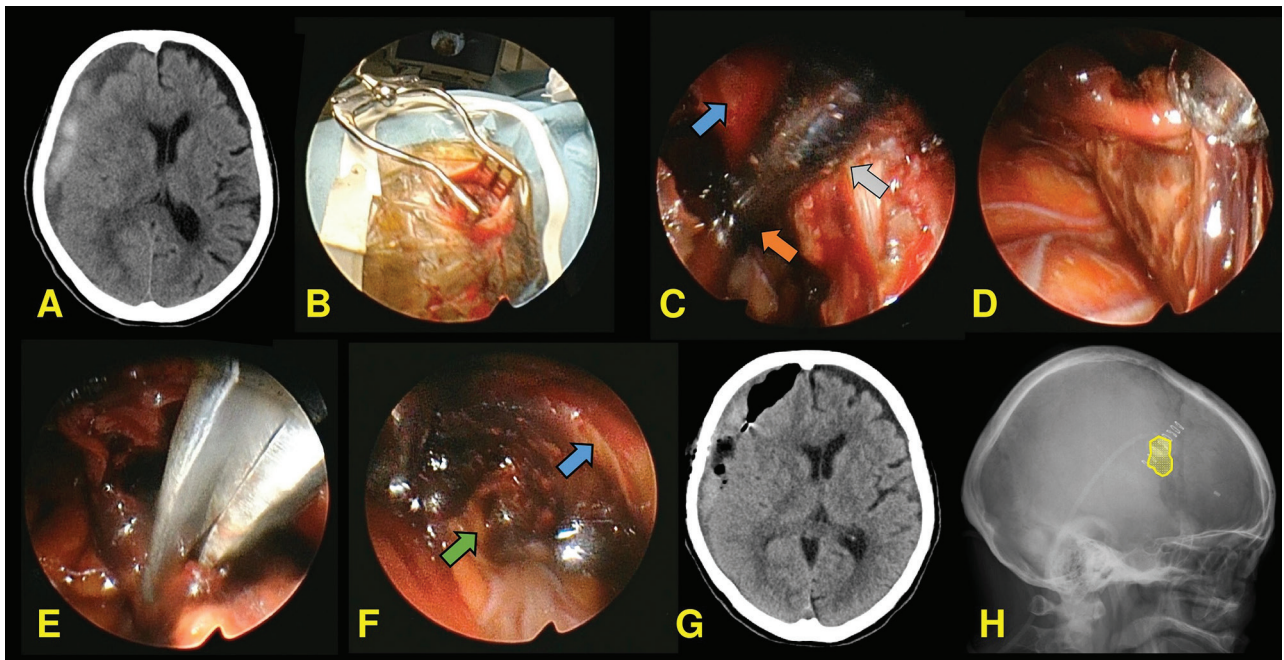


Fig. 2 Preoperative CT image (A). A 3-cm linear skin incision was made parallel to the coronal suture, and a burr hole was made on the point under which the hematoma thickness was largest (B). Rigid hematoma (red arrow in C) was endoscopically evacuated by suction cannula (gray arrow in C). Some of the hematoma was too stiff to be evacuated (D), so we used micro-scissors to remove the hematoma as piecemeal (E). We intentionally left some part of the hard hematoma in the parietal and posterior regions (green arrow in F). As endoscopic findings, the outer membrane seemed red, suggesting inflammation (blue arrows in C, F). The postoperative CT showed sufficient decompression (G) with small craniotomy (H). CT: computed tomography.

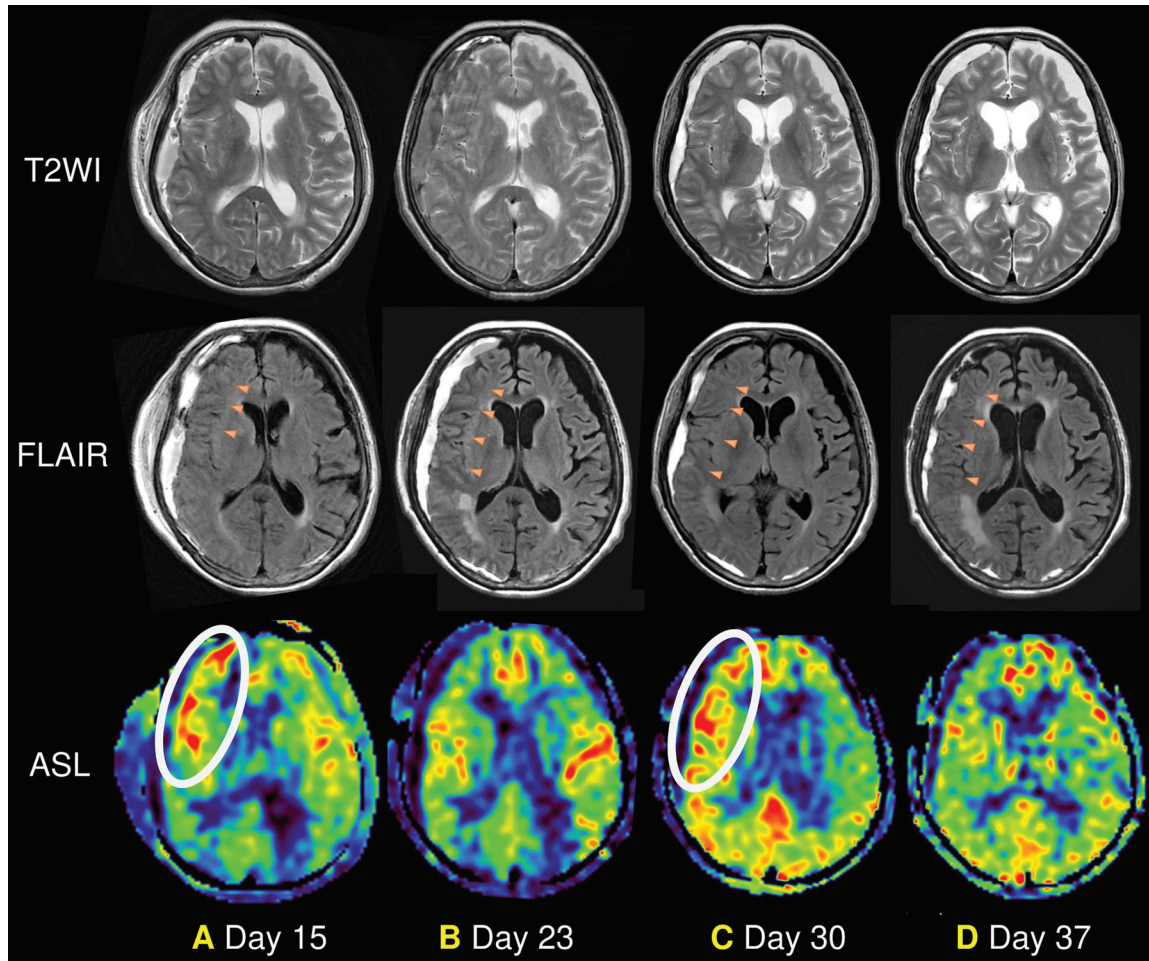


Fig. 3 Postoperative radiological images: On day 15, T2WI and FLAIR images showed no brain edema nor hemorrhage, but SCLI was still observed (red arrowheads in A). ASL image showed CBF increase in the cortex, especially under the removed hematoma (circle in A). On day 23, the SCLI remained in the FLAIR image (red arrowheads in B), but ASL image showed normalized CBF (B). SCLI was consistently observed in the FLAIR images on days 30 and 37 (red arrowheads in C and D). However, CBF easily changed depending on the blood pressure increase (circle in C) and decrease (D). FLAIR: fluid-attenuated inversion recovery, SCLI: subcortical low intensity, T2WI: T2-weighted image.

MMT 1/5. T2WI and FLAIR images showed no brain edema nor hemorrhage, but SCLI was still observed (red arrowheads in Fig. 3A). ASL image showed CBF increase in the cortex, especially under the removed hematoma (circle in Fig. 3A). Although seizures arose as a differential diagnosis, due to the presence of SCLI on FLAIR images,^{8–10} we diagnosed CHP syndrome and strengthened the blood pressure reduction under 130 mmHg with concentrated glycerin administration. We did not administer anticonvulsants considering their side effects. Her paresis gradually improved as MMT 5/5. The SCLI was still observed on the FLAIR image on day 23 (red arrowheads in Fig. 3B), but ASL image showed normalized CBF (Fig. 3B). However, on day 26, she presented oliguria and

heart failure may be due to blood pressure reduction. We made her blood pressure under 160 mmHg. Her oliguria and heart failure gradually improved, and she presented no new neurological deficits. However, the SCLI was still observed on the FLAIR image on day 30 (red arrowheads in Fig. 3C), and ASL image showed CBF increase (circle in Fig. 3C). On day 35, her overall condition had calmed down, so we regulated her blood pressure around 120 mmHg. On day 37, the SCLI was observed on the FLAIR image (red arrowheads in Fig. 3D), but ASL image showed normalized CBF (Fig. 3D). As we described above, consistently after the surgery, the SCLI on the FLAIR image had been observed, and CBF was changed easily depending on the blood pressure in the chronic phase. On day 84, she was discharged

home without any assistance after rehabilitation. As a side note, the diffusion-weighted image and apparent diffusion coefficient map consistently revealed a slightly low-intensity area in the same regions where SCLI was observed on FLAIR images (Supplementary Figs. 1 and 2; Supplementary figures are available online).

Discussion

Subacute subdural hematoma

Traumatic ASDH is a major clinical entity in traumatic brain injury, which usually needs cranioplastic craniotomy or large decompressive craniectomy under general anesthesia.¹²⁾ ASDH patients are recommended to undergo surgical hematoma removal in case of a thickness of hematoma greater than 10 mm or a midline shift greater than 5 mm on CT image, regardless of their Glasgow Coma Scale score.¹³⁾ However, the initial surgical indication is often based on the comprehensive evaluation, including age, comorbidities, and neurological deficits.¹²⁾ Therefore, patients with a large hematoma without neurological deficits are sometimes treated conservatively, and some of them require subacute surgery due to enlarged SASDH.^{2,14–18)} However, the true mechanism of the SASDH development is unknown, and little is also known about hemodynamic changes, the optimum treatment strategies, and perioperative complications.

Mechanism of development of SASDH

The mechanisms of delayed-onset expansion of hematoma were hypothesized to involve an influx of cerebrospinal fluid entering the subdural space, or trans-capillary shift of blood plasma through dilated capillaries as well as petechial hemorrhaging in the neomembrane of the hematoma.²⁾ Recently, the latter hypothesis has been popular that the conversion of ASDH to SASDH is an inflammatory reaction process with a delayed hypersensitivity reaction in the neomembrane with sensitized T lymphocytes.⁴⁾ Regarding chronic SDH (CSDH), the outer membrane is pathologically¹⁹⁾ and endoscopically investigated, and a similar inflammation-related hypothesis is proposed.¹¹⁾ The endoscopically observed outer membrane of CSDH changes from white, red, yellow, and white in order, reflecting the inflammation progression and remission.¹¹⁾ In our SASDH case, we did not pathologically analyze, but the endoscopic findings showed a red outer membrane (blue arrows in Fig. 2C and 2F), which suggested inflammatory progression.¹¹⁾ Our endoscopic findings would support the inflammation-related hypothesis of SASDH conversion.

CBF change in SASDH

In patients with “severe” ASDH, it has been demonstrated that CBF markedly decreases immediately after the injury.^{20,21)} Subsequent to CBF reduction, CHP frequently occurs in the cortex located beneath an evacuated ASDH, and prolongs to be associated with unfavorable outcomes.^{22,23)} Also, in patients with CSDH, asymptomatic or subclinical CHP is postoperatively observed in approximately 50%.^{24,25)} However, regarding SASDH, only one report discussed the CBF changes, and it hypothesized that the hypoperfused tissue in the acute phase might become hyperperfused during the subacute phase owing to impaired autoregulation, and the CHP may be responsible for the development of the SASDH, leading to deterioration.⁶⁾ Whether this hypothesis is correct or not remains unknown due to the small number of cases, but our case also suggested the impaired CBF autoregulation after the long-time brain compression by ASDH.

Our patient's CBF and SCLI changes can be looked back at in three phases: acute, postoperative, and chronic phases. In the acute phase, our case demonstrated CBF increase against the brain compression by the hematoma on day 3 (circle in Fig. 1C), but normalized on day 7 (Fig. 1D). The hematoma did not enlarge or shrink, so the gradual restructuring of microcirculation may have normalized CBF. From the acute to peri- and postoperative phases, SCLI on FLAIR images gradually enhanced and remained (arrowheads in Fig. 1C–1E and 3A–3D), suggesting ischemic change or free radical accumulation.^{7–10)} In the postoperative phase, ASL image on day 15 showed CBF increase in the cortex located beneath an evacuated ASDH (Fig. 3A). This might be due to relief from the compression by ASDH. In addition, we hypothesized that this postoperative CBF increase and CHP syndrome were also due to cortical ischemic change or free radical accumulation owing to the long-time brain compression by ASDH, leading to the CBF dysregulation. From the postoperative to chronic phases, the SCLI on FLAIR images on day 23, 30, and 37 (Fig. 3B–3D) did not change but remained, suggesting an organic change, not a temporary change. In the chronic phase, the CBF is easily upregulated or normalized depending on the blood pressure (Fig. 3C, 3D). This may be because of the CBF dysregulation caused by the organic change due to long-term brain compression. As described above, the mechanism of CBF increase in the three phases may be different. To avoid this postoperative CHP syndrome, easily changeable CBF dynamics, and organic change in the postoperative and chronic

phases, we should have performed surgery in the early stage to resolve brain compression despite the lack of neurological deficits.

Endoscopic hematoma removal for SASDH

Recently, in addition to ASDH²⁶⁾ and CSDH,¹¹⁾ reports about endoscopic hematoma removal with small craniotomy for SASDH have been increasing.^{27–35)} This minimally invasive surgery could address the disadvantages of craniotomy, such as massive blood loss and the side effects of the general anesthesia, especially for the elderly with multiple comorbidities. Also, our endoscopic procedure reduces the operative time as 46 minutes, with a small bleeding amount of 10 mL. SASDH patients usually do not have massive brain contusion leading to brain swelling, but rather hematoma simply compressing the surface of the brain. Besides, the brain does not rapidly expand after hematoma removal, despite severe brain compression. Therefore, we first think that the endoscopic procedure would be a useful method for patients with SASDH. However, we could remove the hematoma only about half by the endoscopic procedure due to hard hematoma while the reported removal rates for ASDH and CSDH were higher than that in this case.^{11,26)} Also, general anesthesia has neuroprotective effects by reducing cerebral edema and intracranial pressure^{36,37)} but we did not use it. Therefore, general anesthesia could have prevented the CHP syndrome by providing neuroprotective effects. The optimum surgical strategy would be different depending on the situation, hematoma stiffness, and patients' status, so it should be further discussed.

Limitations

Seizure arose as a differential diagnosis, but we diagnosed CHP syndrome. This is because (1) we did not use anticonvulsants, but her symptoms improved. (2) SCLI on FLAIR images were observed, suggesting cytotoxic edema⁷⁾ and free radical production,^{8–10)} leading to easily changed CBF. However, a seizure could not be entirely ruled out. We should have performed electroencephalography and should do a careful follow-up to avoid her recurrent seizures.

Conclusions

We showed the endoscopically treated SASDH patient with CBF's chronological changes on ASL images and SCLI on FLAIR images. CHP syndrome after endoscopic treatment for SASDH is a rare complication, but we should be careful. Our

findings suggested that the long-time brain compression would lead to dysfunction of CBF autoregulation.

Ethics

This study was approved by our hospital ethics committee. The patients in our report provided written consent forms.

Acknowledgments

We are thankful to the medical staff for supporting our work and data acquisition.

Conflicts of Interest Disclosure

The authors report no conflicts of interest concerning the materials or methods used in this study, or the findings presented in this paper. No sources of financial or material support were received. This article and content of this study were not published or presented previously.

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