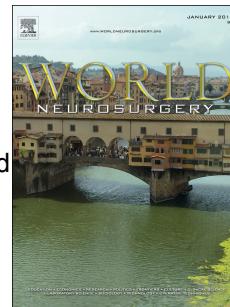


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Prolonged cerebral hyperperfusion and subcortical low intensity on fluid attenuated inversion recovery images: unusual manifestation after removal of organized chronic subdural hematoma

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Key words: organized chronic subdural hematoma, cerebral hyperperfusion, subcortical low

intensity on fluid attenuated inversion recovery images

Abbreviations: CSH, chronic subdural hematoma; CT, computed tomography; FLAIR, fluid attenuated inversion recovery; MRI, magnetic resonance imaging; POD, postoperative day; SCLI, subcortical low intensity; SPECT, single-photon emission computed tomography with N-isopropyl-p-[¹²³I]-iodoamphetamine

ABSTRACT

Background: Cerebral hyperperfusion sometimes occurs after removal of chronic subdural hematoma (CSH) and usually resolves within a few days without any symptoms. Subcortical low intensity (SCLI) on fluid attenuated inversion recovery (FLAIR) magnetic resonance images is rare and has been reported in some diseases other than CSH. The authors describe a case of organized CSH who suffered prolonged neurologic deterioration, SCLI and cerebral hyperperfusion postoperatively.

Case Description: An 81-year-old man, presenting with left hemiparesis, underwent craniotomy for right organized CSH after 2-time burr-hole surgeries. After the craniotomy, the symptom improved, but on postoperative day 2, left hemiparesis, hemispatial neglect and hemisomatagnosia developed. Magnetic resonance imaging revealed SCLI on FLAIR images and single-photon emission computed tomography with *N*-isopropyl-p-[¹²³I]-iodoamphetamine revealed cerebral hyperperfusion in the right hemispheric cortex. Antihypertensive treatment improved the symptoms gradually with improvement of the image findings, which resolved completely one month post-surgery.

Conclusions: The authors first reported a case of organized CSH, which showed postoperative neurologic deterioration, associated with prolonged cerebral hyperperfusion and SCLI on FLAIR images. Prolonged cerebral hyperperfusion could be a cause of postoperative neurologic deterioration in organized CSH.

INTRODUCTION

Organized chronic subdural hematoma (CSH) accounts for 6-19% of CSH.^{1,2} It consists of solid structure with multiseptated hematoma and thick inner and/or outer membranes, and therefore enlarged craniotomy is required to achieve decompression of brain.¹⁻³ Postoperative neurologic deterioration may occur as a result of perioperative complications such as seizure, cerebral infarction or intracerebral hemorrhage.⁴⁻⁶ However, prolonged cerebral hyperperfusion is not well-known as a cause of neurologic deterioration. Moreover, subcortical low intensity (SCLI) on fluid attenuated inversion recovery (FLAIR) magnetic resonance images, which is a rare image finding, has never been reported in CSH.⁷⁻¹⁰ We here report a case of organized CSH who had neurologic deterioration postoperatively associated with prolonged cerebral hyperperfusion and SCLI.

CASE REPORT

An 81-year-old man with a past medical history of prostate cancer presented with mild left hemiparesis. Computed tomography (CT) revealed right CSH, and therefore the patient underwent single burr-hole evacuation of CSH and irrigation of the subdural cavity (Fig. 1). The symptom improved and the patient was discharged in a week. However, 2 weeks after the surgery, symptomatic hematoma regrowth was observed, and the patient underwent the second burr-hole surgery, in which hematoma could not be evacuated because of its organization. Therefore, enlarged craniotomy was performed, which revealed thick outer membrane, organized hematoma with sponge-like structures and paste-like materials, and inner membrane partially adhering to the brain surface (Fig. 2). The membranes and hematoma were removed as much as possible. Postoperatively, systolic blood pressure was controlled below 140 mmHg. The symptom improved, and CT on postoperative day (POD) 1

revealed satisfactory removal of hematoma and right hemisphere re-expansion (Fig. 3). On POD 2 when systolic blood pressure was elevated beyond 170 mmHg, moderate left hemiparesis, hemispatial neglect and hemisomatagnosia developed. Magnetic resonance imaging (MRI) did not reveal acute cerebral infarction or intracerebral hemorrhage but SCLI and sulcal hyperintensity on FLAIR images in the right hemisphere (Fig. 4A). Electroencephalogram revealed neither epileptiform nor seizure patterns. On POD 7, single-photon emission computed tomography with N-isopropyl-p-[¹²³I]-iodoamphetamine (SPECT) revealed cerebral hyperperfusion in the right hemispheric cortex (Fig. 4B). Oral amlodipine (5mg/day) was started, and thereafter the symptoms improved gradually. MRI on POD 14 still revealed SCLI with the appearance of right epidural fluid collection, and SPECT on POD 17 showed mild cerebral hyperperfusion in the right hemispheric cortex (Fig. 4 C and D): around that time, hemispatial neglect and hemisomatagnosia disappeared but mild left hemiparesis continued. No SCLI and cerebral hyperperfusion were shown on MRI and SPECT on POD 31 when all symptoms including left hemiparesis completely resolved, although the right epidural fluid collection increased possibly due to the disappearance of cerebral swelling associated with cerebral hyperperfusion (Fig. 4 E and F). MRI at 5 months postoperation showed neither SCLI nor epidural fluid collection (Fig. 4G).

DISCUSSION

Cerebral hyperperfusion can occur after removal of CSH.¹¹⁻¹³ In this case, we could detect no abnormality on CT, MRI or electroencephalogram other than SCLI and sulcal hyperintensity on FLAIR images when postoperative neurologic deterioration developed; and therefore, we suspected cerebral hyperperfusion, which was confirmed by SPECT. Ogasawara et al.¹¹ reported cerebral hyperperfusion, which lasted for a few days, after

burr-hole surgery for liquid CSH. They also reported cerebral hyperperfusion-induced acute agitated delirium which improved within a day.¹² As to the mechanism of cerebral hyperperfusion, Kuhl et al.¹³ observed hypervolemia in the brain cortex after removal of CSH, which probably represented dilated vessels. Ogasawara et al.¹¹ also suggested that chronic brain compression by CSH might cause dysautoregulation of cerebral blood flow, and therefore even normal perfusion pressure dilated dysregulated cerebral vessels after brain decompression by removal of CSH, leading to cerebral hyperperfusion.

On the other hand, SCLI on FLAIR images has been reported in meningitis, viral encephalitis, leptomeningeal metastasis, spontaneous intracranial hypotension, diffuse axonal injury and moyamoya disease⁷⁻¹⁰: however, there are no reports regarding the association with CSH as far as we know. Although the detailed mechanisms remain unclear, we previously reported the possible relationships between free radical production and SCLI on FLAIR images.¹⁰

In this case, cerebral hyperperfusion lasted for at least 10 days between the first and the second SPECTs associated with neurologic deterioration. The longer duration of compression by organized CSH might delay recovery from disturbed autoregulation of cerebral blood flow. Moreover, we speculate that free radicals might be produced, as suggested by SCLI on FLAIR images, contributing to exacerbation and prolongation of cerebral hyperperfusion. Our patient was highly aged and had chronic ischemic brain with leukoaraiosis (Fig. 4), which might be vulnerable to ischemic brain damages by compression of organized CSH.¹⁴ Profound hypoperfusion and subsequent hyperperfusion by decompression of brain might induce free radical production^{15,16}, causing cerebral hyperperfusion.¹⁷ Although organization might contribute to exacerbation and prolongation of cerebral hyperperfusion through longer compression by the solid structure, it is unclear whether the same phenomena occur in

ordinary cases of liquefied CSH.

Neurologic deterioration developed on POD 2 in this case, although cerebral hyperperfusion is considered likely to occur immediately after surgery. We usually reduce systolic blood pressure lower than 140 mmHg after craniotomy until POD 1 to prevent postoperative hemorrhage. This patient also underwent antihypertensive treatment, which might prevent cerebral hyperperfusion, and neurologic deterioration occurred after discontinuation of the treatment and systolic blood pressure was elevated beyond 170 mmHg. Antihypertensive treatment was effective for postoperative cerebral hyperperfusion and neurologic deterioration in this case. If free radical production might contribute to cerebral hyperperfusion as we speculated, free radical scavenger might also have been effective for it.¹⁷

CONCLUSION

We presented the first case of organized CSH, which suffered prolonged cerebral hyperperfusion and SCLI on FLAIR images postoperatively. Prolonged cerebral hyperperfusion could be a cause of postoperative neurologic deterioration in organized CSH.

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FIGURE LEGENDS

Figure 1. Computed tomography before the first burr-hole evacuation showing right low-density subdural hematoma with internal septations.

Figure 2. Intra-operative findings showing thick outer membrane (arrow) and organized hematoma with sponge-like structures and paste-like materials (double arrow).

Figure 3. Computed tomography after craniotomy showing satisfactory removal of subdural hematoma and right hemisphere re-expansion.

Figure 4. Postoperative fluid attenuated inversion recovery (FLAIR) magnetic resonance images (**A, C, E, G**) and single-photon emission computed tomography with N-isopropyl-p-[¹²³I]-iodoamphetamine (SPECT) (**B, D, F**).

A, on postoperative day (POD) 2, subcortical low intensity (SCLI) and sulcal hyperintensity are shown in the right hemisphere; **B**, on POD 7, remarked cerebral hyperperfusion is shown in the right hemispheric cortex; **C**, on POD 14, the SCLI is attenuated associated with the appearance of right epidural fluid collection; **D**, on POD 17, cerebral hyperperfusion is reduced; **E**, on POD 31, SCLI disappears, although the right epidural fluid collection increases; **F**, on POD 31, cerebral hyperperfusion also disappears; **G**, at 5 months postoperation, both SCLI and epidural fluid collection disappear.

Fig. 1

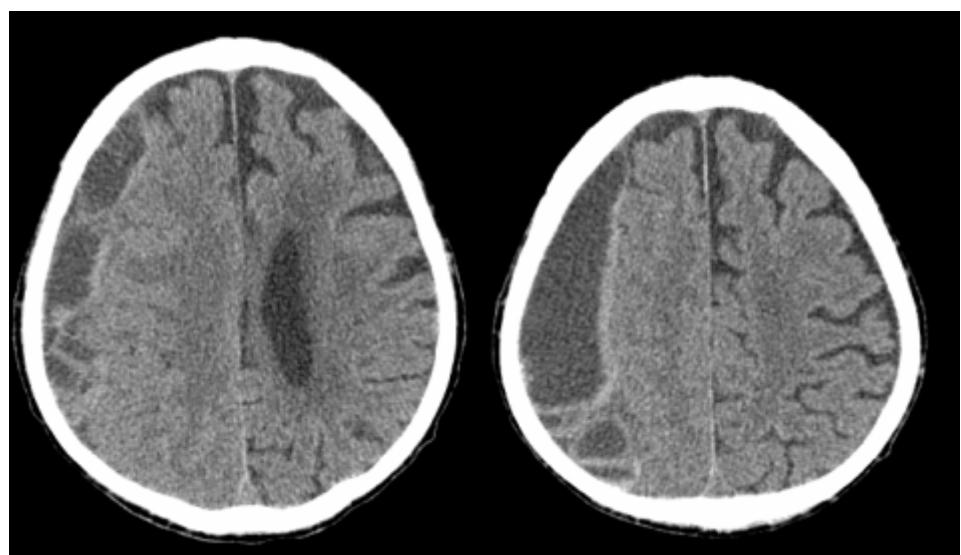


Fig. 2

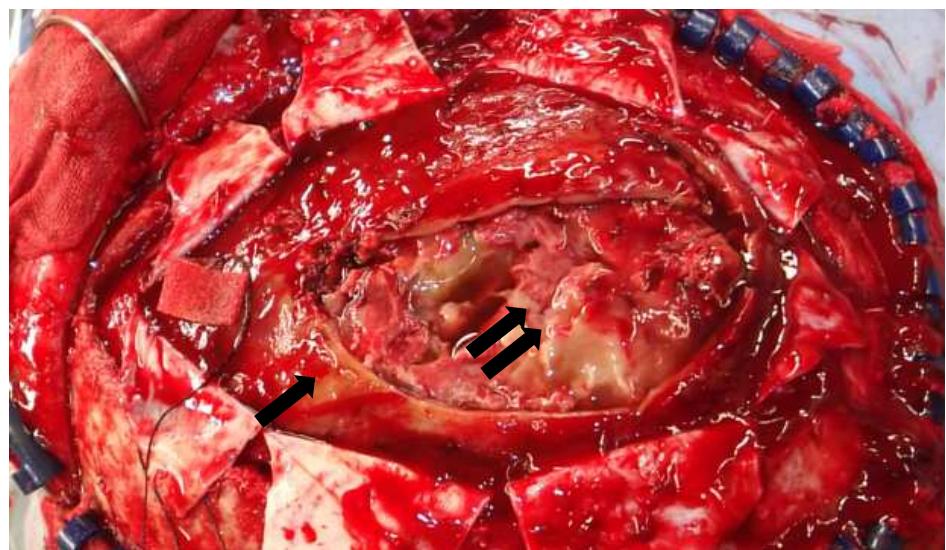


Fig. 3

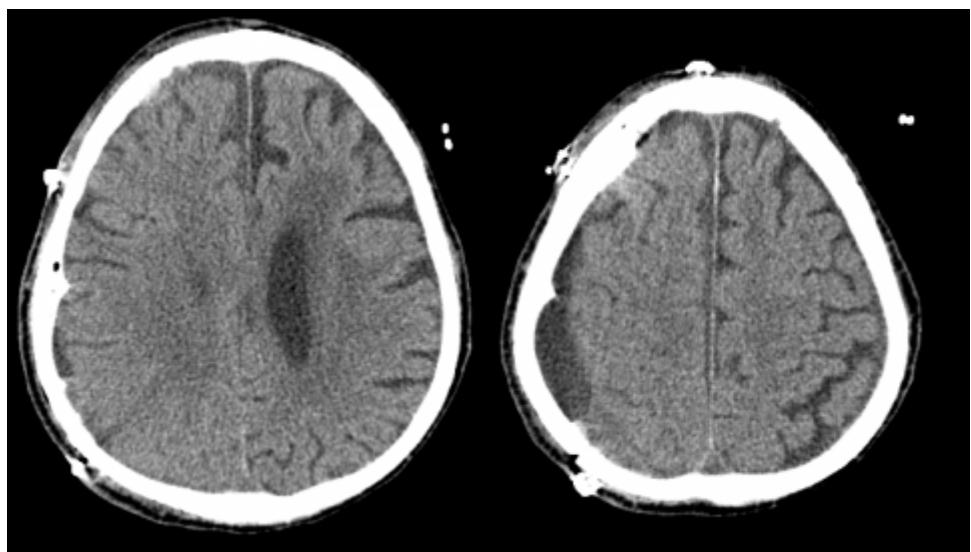
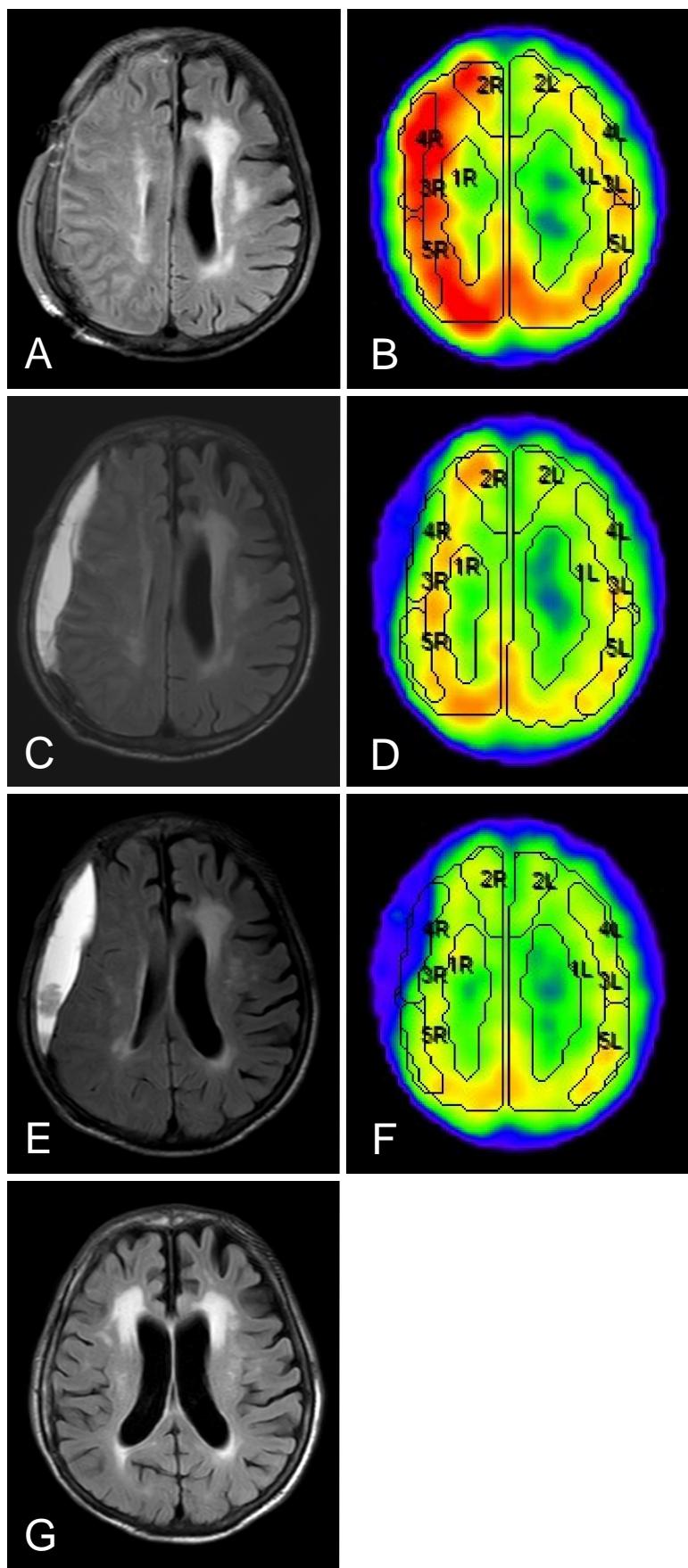


Fig. 4



- Delayed prolonged neurologic deterioration occurred post-surgery for organized CSH.
- SCLI on FLAIR images was observed associated with neurologic deterioration.
- Neurologic deterioration was also associated with prolonged cerebral hyperperfusion.
- SCLI and cerebral hyperperfusion disappeared associated with neurologic improvement.