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*Case Report*

# Hematoma expansion caused by trapped cerebrospinal fluid in subacute phase intracerebral hemorrhage: A case report

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## ABSTRACT

**Background:** Although hematoma expansion (HE) is caused by active bleeding in patients with intracranial hemorrhage in most cases, cerebrospinal fluid (CSF) trapped in the hematoma cavity is not a well-known cause of HE.

**Case Description:** We present a case of subcortical hemorrhage in an 80-year-old woman who experienced neurological deterioration in the subacute phase because of HE caused by CSF pooling in the hematoma cavity. The patient was transferred to our hospital from a previous hospital for surgical treatment because the consciousness disturbance was likely caused by the perihematomal edema that occurred 4 days after onset. Head computed tomography (CT) at admission to our hospital showed a blend sign, and a part of the low-density area of the hematoma was enlarged compared with the CT at admission to the previous hospital. Although the hematoma was located adjacent to the lateral ventricle, no intraventricular hemorrhage was observed. Emergent hematoma evacuation was performed, and intraoperative findings indicated that the enlarged hematoma cavity was caused by CSF pooling. The patient's postoperative course was uneventful. She was transferred to a rehabilitation hospital 16 days after admission to our hospital.

**Conclusion:** Hematomas adjacent to the ventricle and showing a blend sign can expand in the subacute phase because of the trapped CSF.

**Keywords:** Blend sign, Cerebrospinal fluid, Hematoma expansion, Intracerebral hemorrhage, Perihematomal edema

## INTRODUCTION

Hematoma expansion (HE) is independently associated with neurological deterioration in patients with intracranial hemorrhage (ICH) both in the hyperacute and acute phases.[4] In the subacute phase (1–3 days from onset), perihematomal edema and fever are independently associated with neurological deterioration.[4] We present a case of neurological deterioration caused by delayed HE, which was, in turn, due to trapped cerebrospinal fluid (CSF) 4 days after the onset. To the best of our knowledge, this is the first report of HE, caused by trapped CSF, mimicking a perihematomal edema.

## CASE DESCRIPTION

An 80-year-old woman with a history of cerebral infarction and hyperlipidemia complained of a sudden visual field defect and hemiparesis without disturbance of consciousness, and was hospitalized.

Head computed tomography (CT) performed 1.5 h from onset showed subcortical hemorrhage in the left occipital lobe. The patient received antihypertensive therapy. Four days after admission, her consciousness level decreased and she underwent a head CT. She was diagnosed with perihematomal edema and considered for hematoma evacuation and decompressive craniotomy. Subsequently, the patient was transferred to our hospital for further treatment. Neurological examination revealed consciousness disturbance, anisocoria, and right hemiparesis. She scored 5 points on the Glasgow Coma Scale (E1M3V1). Head CT during admission to our hospital [Figure 1a] showed a low-density area (LDA) enlargement, niveau formation in the hematoma cavity, and uncal herniation with worsened midline shift, compared with the head CT taken during admission at the previous hospital [Figure 1b]. LDA CT values ventral to the hematoma at the time of admission to the previous hospital and our hospital were 19.32 Hounsfield units (HU) and 24.40 HU, respectively [Figures 1a and b]. Hematoma CT values at the time of admission to the previous hospital and our hospital were 67.60 HU and 72.05 HU, respectively [Figures 1a and b]. Emergent craniotomy was performed for hematoma evacuation. A hard clot (first observed in the occipital lobe after corticotomy) was removed, which revealed an aspirable fluid-containing blood [Figure 2a]. An aspirable soft clot was present at the bottom of the hematoma cavity [Figure 2b], beyond which a thin ventricular wall with a small hole was observed, through which the CSF entered the cavity [Figure 2c]. Although postoperative CT showed residual hemorrhage, an improved midline shift was also observed, and the uncal herniation had disappeared [Figure 2d]. The consciousness disturbance in the patient was resolved, and the right hemiparesis improved. The patient was transferred to a rehabilitation hospital (modified Rankin scale [mRS] score: 4) after a 16-day stay at our hospital, and her mRS score at 8 months from onset was 1.

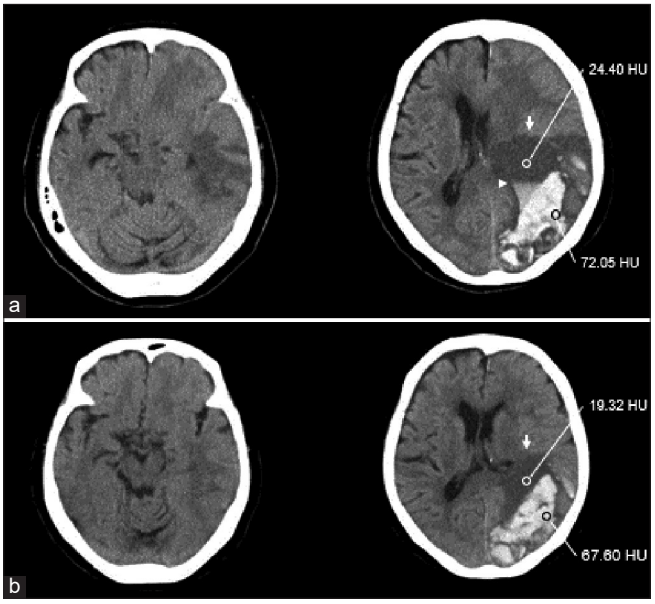


**Figure 1:** (a) Computed tomography (CT) at admission to our hospital shows an enlargement of hematoma cavity with a worsened midline shift and uncal herniation. In spite of no hematoma growth, a part of the Low-density area (LDA) (arrow) with niveau formation (arrow head) in the hematoma cavity was enlarged. LDA and high-density area (HDA) CT values were 24.40 Hounsfield Unit (HU) and 72.05 HU, respectively. (b) CT at admission to a previous hospital shows subcortical hemorrhage at the occipitotemporal lobe with blend sign and no uncal herniation and intraventricular hemorrhage. LDA (arrow) and HDA CT values were 19.32 HU and 67.60 HU, respectively.

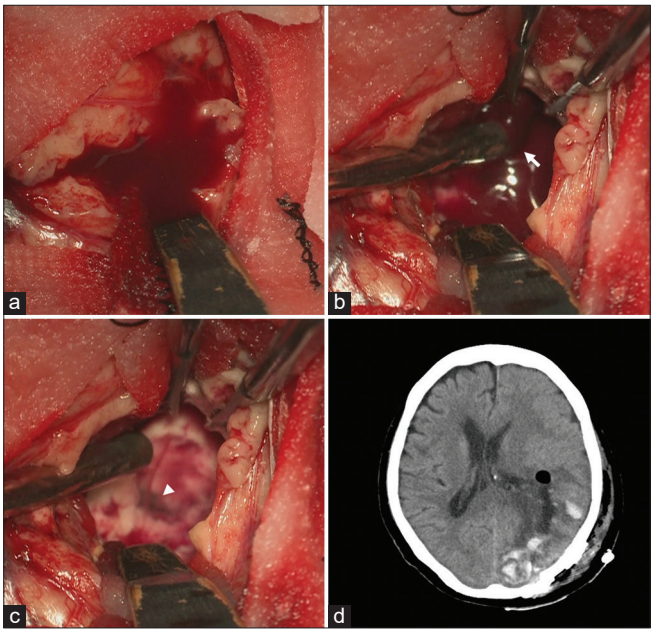


**Figure 2:** (a) Fluid-containing hemorrhage after hard clot removal. (b) Aspirable soft clot (arrow) at the bottom of the hematoma cavity. (c) CSF entering into the hematoma cavity through ventricular wall hole (arrowhead). (d) Postoperative CT.

## DISCUSSION

Herein, CT and intraoperative findings indicated that increased CSF components in the hematoma cavity led to HE and neurological deterioration. CSF, brain white matter, and hematoma CT values were 0–15 HU, <40 HU, and <80 HU, respectively.[2] In traumatic brain injury patients with diffuse brain swelling, the brain edema CT values range from 11 to 24 HU.[2,5] Although the LDA adjacent to the hematoma

mimicked a perihematomal edema, the LDA on CT taken 1.5 h from the onset did not appear the same. Intraoperative findings revealed that the LDA component ventral to the hematoma was a fluid-containing hematoma, with a LDA CT value higher than that of CSF, but lower than that of the hematoma. Based on the finding that CSF entered the hematoma cavity through a tiny hole in the thin ventricular wall, we concluded that the size of the enlarged LDA was not caused by worsening brain edema, but by enlargement of the hematoma cavity caused by an increased CSF component.

A CT blend sign is reportedly associated with HE. Indeed, in our case, CT performed on admission in the previous hospital showed a blend sign-like appearance: (1) blending of hypoattenuating and hyperattenuating regions with apparent margins between the two regions in the hematoma cavity; (2) >18 HU difference between the hypoattenuating and hyperattenuating regions in the hematoma; and (3) a hypoattenuating region not encapsulated in the hyperattenuating region.[3] The mechanism of formation of the blend sign was by the blood plasma, which showed a hypoattenuating region due to active bleeding.[3] In some patients, when the hematoma is located adjacent to the ventricular and/or subarachnoid space, the blend sign might reflect CSF pooling in the hematoma cavity rather than active bleeding. Although preoperative CT did not show intraventricular hemorrhage (IVH), there was communication between the ventricular and hematoma cavities in this case. In addition, we found a thin lateral ventricle wall with a tiny hole at the bottom of the hematoma cavity, in the present case. When the hemorrhage occurred, the hematoma could not make a large enough hole in the ventricle wall to cause IVH, and the clot covered the tiny hole in the hyperacute and acute phases. The clot might have melted with time and its adhesion might have weakened, functioning like a "check valve," and the CSF was able to move into the hematoma cavity from the lateral ventricle, but not vice versa. The high colloidal osmotic pressure of subdural hematoma is reported to be a cause of chronic subdural hematoma enlargement.[1] Thin ventricle walls with tiny holes might act as a semipermeable membrane, and the CSF might move into the hematoma cavity due to the high osmotic pressure caused by the hematoma.

In general, HE caused by ICH occurs in the hyperacute phase. However, in cases of ICH adjacent to the ventricle, with a blend sign or trapped CSF, HE may occur in the subacute phase. Therefore, careful observation extending into the subacute phase is required, and the patients and their families need to be informed of this possibility. In addition, although we performed craniotomy hematoma evacuation with general anesthesia in the present case, stereotactic or endoscopic hematoma evacuation with local anesthesia, which are less invasive than craniotomy hematoma evacuation, might be considerable in ICH cases with trapped CSF as the main component of HE is estimated to be aspirable fluid-containing soft clot.

## CONCLUSION

ICH cases with a CT blend sign are potential cases of hematoma with CSF, especially when the hematomas are adjacent to the ventricle without IVH. Careful observation is required, as in these cases, HE might often occur in the subacute phase.

### 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.

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