Brain Stem Hemorrhage Following Burr Hole Drainage for Chronic Subdural Hematoma
—Case Report—

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Abstract
A 76-year-old man presented with brain stem hemorrhage after burr-hole drainage for bilateral chronic subdural hematomas. Neuroimaging demonstrated resolution of the transtentorial herniation but also detected new brain stem hemorrhage, manifesting as gait disturbance. He recovered after conservative treatment. Asymmetrical and rapid decompression, which leads to vascular disruption and/or sudden increase in cerebral blood flow, was probably responsible for the secondary brain stem hemorrhage. Therefore, simultaneous and bilateral decompression with a slow rate of evacuation of massive bilateral chronic subdural hematomas is recommended to prevent serious complications such as secondary intracranial hematoma.

Key words: brain stem, chronic subdural hematoma, decompression, postoperative hemorrhage

Introduction
Chronic subdural hematoma (CSDH), which commonly affects the elderly, is the most benign form of intracranial hematoma with a mortality rate ranging from 0.5 to 4.0%.[7,16,19] Burr hole drainage is the most commonly performed treatment modality for evacuation of a CSDH, and is known to be a safe method with a low morbidity rate of 0–9%. However, postoperative complications, including cerebral edema, hematoma re-accumulation, subdural empyema, tension pneumocephalus, and intracranial hemorrhage in other sites, have occasionally been reported.[2,11,18,24,25] Intracranial hematomas following burr hole drainage may occur either close to or remote from the original site, but hemorrhage in the brain stem secondary to the surgery is extremely rare.[2,4,8,14] We treated a 76-year-old man who suffered brain stem hemorrhage after bilateral CSDH evacuation, who fortunately recovered with only mild gait disturbance.

Case Report
A 76-year-old man was admitted with a slowly diminishing level of consciousness over a 2-week period. Neurological examination revealed stuporous mental status without definite motor weakness. The patient had no medical history of arterial hypertension or significant head trauma. Laboratory studies, including clotting profiles and liver function tests, did not reveal any abnormality. Computed tomography (CT) showed large bilateral subdural hematomas with fluid levels compressing the cerebral hemispheres (Fig. 1). The cortical sulci were not visualized, and the bilateral medial temporal lobes were displaced inferomedially to the cerebellopontine angle cistern, suggesting transtentorial herniation.

Emergency removal of the bilateral hematomas was performed under general anesthesia. Bilateral burr holes were made consecutively, and the dural surfaces were exposed at the same time. The dura and outer membrane on the left side were opened first, followed by the right side about one minute later. Opening of the outer membranes allowed the dark brown hematomas to gush out via the openings. We tried to evacuate the hematomas gradually, but rapid decompression occurred during the initial period of the procedure due to the high pressures within the subdural hematomas. Thereafter, closed-system subdural drainage catheters were inserted through each burr hole. The patient’s vital signs were stable during the operation, and no elevation of arterial blood pressure was observed. His consciousness improved on the 1st postoperative day. However, as the patient started to mobilize on the 2nd postoperative day, he complained of gait disturbance with mild lower limb weakness.

CT and magnetic resonance (MR) imaging demonstrated resolution of the transtentorial herniation, but also detected brain stem hemorrhage not present on the preoperative images (Figs. 2 and 3A). During the postoperative period, his blood pressure was maintained within the normotensive range. The patient remained stable and was managed conservatively. Follow-up MR imaging 7 days after the operation revealed a subacute hematoma in the brain stem without evidence of vascular malformation.
Fig. 1 Preoperative computed tomography scans showing thick bilateral chronic subdural hematomas with fluid levels. The bilateral medial temporal lobes are displaced inferomedially to the cerebellopontine angle cistern, suggesting transtentorial herniation.

Fig. 2 Computed tomography scans obtained 2 days after burr hole evacuation of the subdural hematomas demonstrating resolution with restoration of the transtentorial herniation. However, brain stem hemorrhage was also observed not seen on the preoperative images.

and minimal subdural fluid collection around the bilateral cerebral hemispheres (Fig. 3B). His lower limb weakness gradually improved, and he became ambulatory and was discharged 14 days after admission.

**Discussion**

Only two cases of brain stem hemorrhage as a complication of surgery for CSDH have been reported, and both cases were detected at autopsy.\(^{10,17}\) The present patient survived the brain stem hemorrhage that developed after the evacuation of bilateral CSDHs.

The principal mechanism causing postoperative intracerebral hemorrhage is a sudden increase in cerebral blood flow combined with defective vascular autoregulation.\(^{2,12}\) However, the pathophysiology of brain stem hemorrhage following intracranial hematoma decompression has not been determined. Brain stem hemorrhage is often the cause of immediate death in patients, so the hemodynamic changes are difficult to study. A previous animal study investigated the mechanism of brain stem hemorrhage secondary to supratentorial decompression, and suggested that the brain stem hemorrhage was likely due to damaged vessels caused by increased intracranial pressure (ICP).\(^6\) Rapid alleviation of increased ICP, under conditions of elevated blood pressure, could disrupt the small injured vessels, invariably resulting in brain stem hemorrhage.

In the present case, preoperative CT showed transtentorial herniation was present, indicating that the blood vessels near the brain stem might be stretched and distorted.\(^{2,15}\) The left-sided decompression was performed with short time lag before the right side procedure, which under such conditions might have led to transient aggravation of the right-sided transtentorial herniation by shifting and displacing the brain stem. Consequently, the vessels surrounding the brain stem may have torn, precipitating the hemorrhage into the brain stem. Although the time interval between the procedures was short, about one minute, vascular disruption could occur easily since the transtentorial herniation was already present, and the vessels surrounding the brain stem had already been maximally stretched. In fact, cranial nerve paresis and brain stem dysfunction have been reported after the removal of bilateral CSDHs,\(^{13}\) suggesting that the complications may be due to rapid unilateral decompression, which leads to sudden distortion of the midline structures and induces transient contralateral transtentorial herniation.

Single photon emission computed tomography has demonstrated that cerebral blood flow in patients with CSDHs is diminished, particularly in the ipsilateral basal ganglia and thalamus,\(^3\) followed postoperatively by progressive normalization of blood flow.\(^23\) The brain stem blood flow in the present patient might have been reduced...
by the severe compression because of the massive bilateral subdural hematomas, but no blood flow study was performed. Physiological aging of the cerebral vascular tree is associated with poor tolerance of sudden variations in cerebral blood flow.\textsuperscript{20} Considering that the present patient was elderly, the increased fragility of the small blood vessels might not have been able to sustain the rapid changes in the brain stem blood flow during decompression of the subdural hematoma. Consequently, the brain stem hemorrhage in the present case was likely due to the mechanical disruption of small fragile vessels caused by transient aggravation of the transtentorial herniation during the first decompression procedure, and accentuated by an increase in blood flow caused by breakthrough-like dysregulation triggered by the abrupt supratentorial decompression. Other factors responsible for brain stem hemorrhage, such as vascular malformations, bleeding tendency, and perioperative hypertension,\textsuperscript{2,21} could be ruled out based on the patient’s medical records and the findings of postoperative MR imaging.

The surgical treatment for bilateral CSDHs is not different from that for unilateral CSDH, and includes evacuation through a twist drill hole, burr hole, or craniotomy. Gradual decompression avoiding rapid changes in blood flow may be accomplished by primary drainage through a frontal burr hole and/or then applying cotton to the burr hole immediately after opening the outer membrane. Bilateral simultaneous decompression is also helpful if excessive drainage through burr holes can be controlled, which can prevent the brain parenchyma from abruptly shifting. Continuous closed-system catheter drainage has been proposed for the treatment of CSDH, as this allows the brain to re-expand slowly to obliterate the subdural space.\textsuperscript{1,22} This method might be most useful for the treatment of very large bilateral CSDHs that should be decompressed gradually and symmetrically.

The present case of brain stem hemorrhage following the evacuation of bilateral CSDHs indicates that asymmetrical and rapid decompression, which leads to vascular disruption and sudden increase in cerebral blood flow, may have caused this complication. Therefore, simultaneous and bilateral decompression with a slow rate of evacuation is recommended for the removal of massive bilateral CSDHs, to avoid serious complications such as secondary intracranial hematoma.

References

13) Okuchi K, Fujio M, Maeda Y, Kagoshima T, Sakaki T:


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Neurol Med Chir (Tokyo) 49, December, 2009