"Contralateral" Acute Subdural and Intracerebral Hemorrhage Occurring Simultaneously after Evacuation of Huge Chronic Subdural Hematoma

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We report an uncommon case of "contralateral" acute subdural hematoma (SDH) and intracerebral hemorrhage (ICH) developing simultaneously after evacuation of a huge chronic SDH associated with previous ischemic stroke. An 80-year-old female admitted with bilateral chronic SDH with midline shift by the left-sided dominant huge hematoma. The patient underwent burr-hole evacuation for the bilateral hematoma. The left-sided huge hematoma was first evacuated and the right-sided small hematoma was drained later. Immediate computerized tomography (CT) scans after surgery revealed acute brain shift contrary to the initial scan and acute SDH and ICH developing contralaterally for the huge hematoma. The pathophysiological mechanisms were discussed. (J Kor Neurotraumatol Soc 2011;7:39-42)

KEY WORDS: Acute subdural hematoma · Chronic subdural hematoma · Intracerebral hemorrhage · Complication.

Introduction

Although the neurological procedure for chronic subdural hematoma (SDH) is largely successful, unexpected neurological deterioration may occasionally impair the postoperative course. Intracerebral hemorrhage (ICH) is the most feared complication after removal of chronic SDH,^{2,5,6,7,9,11} which always develop characteristically on the ipsilateral side to the SDH.⁹⁾ "Contralateral" ICH occurring after evacuation of chronic SDH is an uncommon event. We report an uncommon case of "contralateral" acute SDH and ICH simultaneously developing by acute brain shift after evacuation of a huge chronic SDH associated with previous ischemic stroke.

Case Report

An 80-year-old woman presented with an altered level of consciousness one day ago. She had a history of right hemi-

Received: March 14, 2011 / Revised: March 17, 2011 Accepted: April 6, 2011 Address for correspondence: Cheol Su Jwa, MD Department of Neurosurgery, National Medical Center, 243 Euljiro, Jung-gu, Seoul 100-799, Korea Tel: +82-2-2260-7279, Fax: +82-2-2262-4869 E-mail: chsjwa@hanmail.net paresis (grade IV/IV) after ischemic stroke for about ten years, but she has taken no anti-platelet agents. She had sustained a minor head trauma about a month before admission. On admission, vital signs were within normal limit. Neurological examinations showed drowsy consciousness, pupils of equal size with normal light reaction and right hemiparesis (grade II/III). According to the Glasgow Coma Scale, she was rated 14. Initial computerized tomography (CT) scans showed bilateral chronic SDH with the left-sided dominant huge hematoma, which causes significant mass effect and midline shift from left to right side (Figure 1). CT angiography showed total occlusion of proximal portion of the left middle cerebral artery and severe atherosclerotic changes of cavernous portion of the both carotid arteries. The routine laboratory tests showed severe anemia (6.5 g/dL). We planned evacuation of the chronic SDH after red cell transfusion. She was transfused 2 pints of packed red cell. However, she rapidly deteriorated to stuporous state with fixed pupils. Follow-up CT scans 6 hours after admission showed no significant interval changes compared with initial CT scans.

She underwent emergent evacuation of the SDHs after burr-hole trephination under general anesthesia. First, the left-sided huge hematoma was evacuated. Chocolate-colored dark fluid gushed out. The subdural space was copiContralateral Hemorrhages after Evacuation of Chronic SDH



FIGURE 1. Preoperative computerized tomography (CT) scans show bilateral chronic subdural hematoma with the left-sided dominant huge hematoma, which causes mass effect and midline shift from left to right side.



FIGURE 2. Postoperative computerized tomography (CT) scans just after surgery reveal acute brain shift from right to left side contrary to initial CT scans and acute subdural and intracerebral hemorrhage on the "contralateral" side of the huge subdural hematoma.

ously washed with 0.9% saline until the return was clear and the drainage catheters were inserted subdurally. The right-sided small hematoma was drained later. During surgery for the right-sided small hematoma, there was a sudden copious loss of hematoma fluid (about 100 mL) from the left-sided drainage catheters positioned just at the base level of head. CT scans just after surgery revealed acute brain shift from right to left side contrary to the initial CT scans and acute SDH and subcortical ICH on the contralateral side of the huge SDH (Figure 2). The left-sided drainage catheter was immediately clamped and she has conservatively treated. Follow-up CT scans showed gradual improvement of midline shift. She improved progressively and was able to communicate 3 days after surgery. Two weeks later, she was discharged with clear mentality and improved motor function (grade IV/IV). Followup CT scans 2 months after surgery showed total hematoma resolution state and extensive cerebromalatic change in the territory of the left middle cerebral artery (Figure 3).

Discussion

Huge chronic SDH associated with previous ischemic

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FIGURE 3. Computerized tomography (CT) scan 2 months after surgery shows total hematoma resolution state and extensive cerebromalatic change in the territory of the left middle cerebral artery.

stroke is an unusual presentation to many neurosurgeons. Few cases have rarely been encountered in the English literature.¹⁵⁾ Only one instance of huge chronic SDH mimicking cerebral infarction on CT scans has been observed; moreover, poor outcome after craniotomy has also been documented. The exact mechanism, natural course and surgical complications of huge chronic SDH are still unclear. The decrease of elastance in the brain tissue after ischemic stroke in addition to physiological atrophy may permit chronic SDH to expand unexpectedly.¹⁵⁾. The prexisting deficits may mask neurological deterioration and may allow hematoma growth.

Burr-hole craniotomy is the safest and most effective technique for evacuation of chronic SDH.²⁰⁾ However, this procedure may be occasionally complicated by postoperative hemorrhages. ICH^{2,8,9,11,14,16} or acute SDH^{3,10,17,19} after surgery of chronic SDH or hygroma are very rare, but potentially fatal complications. There have been reports of more than about 30 cases with ICHs after removal of chronic SDH,14) which always occurred characteristically on the ipsilateral side to the chronic SDH if unilateral.¹⁴⁾ "Contralateral" acute SDH by acute brain shift after removal of chronic SDH is an extremely rare complication. Only a few cases of "contralateral" acute SDH following surgery of chronic SDH or hygroma were reported.^{3,10,17,19)} To our knowledge, this is an extremely rare case of "contralateral" acute SDH and ICH developing simultaneously after evacuation of a huge chronic SDH associated with previous ischemic stroke.

The pathophysiological mechanism of "contralateral" hemorrhages after evacuation of chronic SDH is not clear. Insufficient hemostasis at burr-hole site may be a cause of contralateral acute SDH after evacuation of bilateral chronic SDH.⁷⁾ However, in this case, inadequate hemostasis is unlikely as the operative site bleeding was meticulously controlled. In our operative finding, a sudden copious drainage of hematoma fluid during contralateral surgery could have led to perioperative brain shift, which can tear the contralateral bridging veins^{1,3,6,19)} and subsequent "contralateral" acute SDH. ICH after evacuation of chronic SDH may be caused by a sudden increase of cerebral blood flow following rapid decompression, combined with defective auto-regulation, hemorrhage into missing areas of contusion, fragile cerebral vessels, and labile hypertension.^{8,9,11,13,14,16)} Xenon-enhanced CT studies have demonstrated that cerebral blood flow in patients with chronic SDH is decreased, particularly the ipsilateral frontal region,⁵⁾ rolandic cortical area,⁴⁾ putamen and thalamus.^{5,18)} This is restored postoperatively by progressive normalization of blood flow in these regions.^{12,14,18)} In this study, ICH into missing areas of contusion and hypertension are unlikely, as a preoperative CT scans failed to reveal contusion, and there was no elevation of systolic blood pressure during the peri-operative period. The most probable mechanism is a sudden increase of cerebral blood flow in the contralateral frontal region, combined with defective autoregulation. Contralateral fragile vessels due to atherosclerotic change instead of ipsilateral occlusive middle cerebral artery as showed in CT angiography could have not been tolerated during acute brain shift following sudden decompression of a huge chronic SDH.¹⁴⁾

Conclusion

"Contralateral" acute SDH and ICH by acute brain shift must be considered as a possible complication after evacuation of a huge chronic SDH. We stress that sudden decompression should be avoided during surgery for patients with chronic SDH who show evidence of brain compression by large hematoma on imaging. Particularly for the severe cerebral atrophic cases following ischemic stroke, the patient should be monitored carefully and postoperative CT scans should be taken early to look out for possible complications.

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