Massive Intracerebral Hemorrhage Following Drainage of Subdural Hygroma

Sung Soo Kim, M.D., Choong Hyun Kim, M.D., Jin Hwan Cheong, M.D., Jae Min Kim, M.D.
Department of Neurosurgery, Hanyang University Guri Hospital, Hanyang University College of Medicine, Guri, Korea

Subdural hygromas are easily treated by trephination and drainage. Therefore, most neurosurgeons do not consider subdural hygromas seriously. However, various complications including intracerebral hemorrhage may develop after rapid drainage of subdural hygroma although rare. Postoperative intracerebral hemorrhage presents with a rapid deterioration of consciousness and focal neurological deficits occurring immediately after drainage of the subdural hygroma. The authors present an unfortunate massive intracerebral hemorrhage and pneumocephalus following drainage of the bifrontal subdural hygroma. The patient subsequently died. To prevent this disastrous complication, close neurosurgical observation and gradual drainage under a closed system seem mandatory. Possible pathogenic mechanisms for this unfavorable complication is discussed with a review of pertinent literatures.

KEY WORDS: Subdural hygroma · Intracerebral hemorrhage · Pneumocephalus · Drainage.

Introduction

Subdural hygromas are characterized by a subdural collection of cerebrospinal fluid (CSF). Although its pathogenic mechanism is uncertain, it develops by the tearing of the arachnoid membrane and subsequent accumulation of CSF in the subdural space. The surgical procedures for subdural hygromas include drainage through burr holes or twist drills, subdureoperitoneal shunt, and craniotomy. Reaccumulation of fluid, infection, seizure, and hydrocephalus are common complications after surgery. The incidence of intracerebral hemorrhage after drainage of the subdural hygroma is as low as 1 to 5%. This report presents a patient who had rapid neurological deterioration caused by massive intracerebral hemorrhage after drainage of the subdural hygroma.

Case Report

A 83-year-old man was transferred from the orthopedic surgery department because of alteration of consciousness. He was admitted because of an intertrochanteric fracture of the femur that developed after slipping two weeks prior to admission. Other than drowsy mental state, patient's symptoms were unremarkable. He had a history of a mild head injury caused by a fall, but it was not evaluated on admission. Brain computed tomography (CT) scans revealed subdural fluid collected on both frontal regions (Fig. 1). The coagulation parameters including prothrombin time, partial thromboplastin time, and platelet count were normal. Drainage under a closed system was performed through bifrontal burr holes. There was no improvement postoperatively. Follow-up brain CT scans taken on the third postoperative day revealed extensive pneumocephalus, small amounts of subdural collection, and severely compressed brain parenchyma (Fig. 2).
Five days later, his neurological status deteriorated markedly. Additional brain CT scans displayed a huge intracerebral hemorrhage in both frontal areas, severe brain edema, and midline shift to the left side (Fig. 3). Emergency decompressive surgery was planned, but his family refused the operation because of the patient's old age and profound neurological deficits. The patient subsequently died on the seventh postoperative day.

Discussion

The subdural hygromas are not uncommon in the neurosurgical practice. It is one of the various terms used to describe subdural fluid collection such as effusion, benign subdural collection, and external hydrocephalus. The causes of subdural hygroma include surgical procedures, head injuries, infections, congenital malformations, and cranioccephalic disproportions. In the case of traumatic subdural hygromas, the widely accepted pathogenic mechanisms are the tearing of arachnoid membrane and ball-valve hypothesis. Traumatic rupture of the arachnoid membrane results in consequent CSF leakage without reabsorption.

The arachnoid membranes in the sylvian and chiasmatic regions are likely to tear following trauma. Tearing of the arachnoid membrane may act as a ball-valve device. This may prevent the restoration of cohesion within the dura-arachnoid interface layer and can trap a significant amount of CSF.

In the management of subdural fluid collection, serial brain CT scans give important information such as persistence or increase of subdural collection, extent of midline shifting, and ventricular compression. Such information helps to decide the choice of treatment. Commonly used surgical procedures are burr hole drainage with a closed system, twist drill craniostomy with or without a closed system drainage, shunting of the subdural space, and craniotomy. Recollection of subdural fluid collection, infection, seizure, hydrocephalus, and failure of brain expansion are all related complications. However, postoperative intracerebral hemorrhage is rare. Modesti et al. reported 7 (5%) cases of postoperative intracerebral hemorrhage among 140 cases of chronic subdural fluid collections treated surgically. Richter et al. reported one case of mortality by intracerebral hemorrhage among 120 cases of chronic subdural hematoma.

As seen in relevant scientific literatures, a possible pathogenic mechanism for this phenomenon may be a sudden increase in cerebral blood flow combines with faulty autoregulation, and damage to parenchymal vessels secondary to the rapid intraoperative or postoperative shift of the intracranial contents. This mechanism is supported by several pathologic findings. Focal cerebral edema beneath the compressed surface of the brain by impedance of venous drainage causes a decrease in cerebral blood flow in the affected hemisphere. Chronic dilatation of small arterial vessels and loss of carbon dioxide reactivity in the ischemic hemisphere also contribute to the pathogenesis.

To avoid this catastrophic complication, gradual drainage under a closed system is mandatory. Several authors proposed that a closed drainage through a twist drill hole is the safest and most effective surgical technique. This procedure is effective in slow and complete decompression with gradual re-expansion of the brain. It also avoids rapid dynamic intracranial changes.

Although a postoperative massive intracerebral hemorrhage after drainage of the subdural hygroma is a rare complication, it may occur as in this case. It is not easy to predict the possibility of a postoperative intracerebral hemorrhage easily especially when the patient's coagulation parameters are normal. When such a complication is suspected, an immediate brain CT scan should be conducted and surgical removal of the hematoma should be followed.

Conclusion

We report an unusual case of a massive intracerebral hemorrhage following drainage of a subdural hygroma. In patients who undergo a drainage of the subdural hygroma that results in deteriorating neurological conditions, the possibility of an intracerebral hemorrhage should be considered. To prevent this complication, close neurological observation and a slow drainage system seem mandatory.
References