Isolated Intraventricular Hemorrhage Following Evacuation of Chronic-Subacute Subdural Hemorrhage: A Case Report

Abstract
Chronic subdural hemorrhage is commonly encountered in the elderly. However, the outcomes of surgery for chronic subdural hemorrhage are frequently benign, surgeons may sometimes encounter unexpected complications after evacuation of chronic subdural hemorrhage such as isolated intraventricular hemorrhage. A few hypotheses have been developed to explain this phenomenon. Here, we present a case report and explain this phenomenon with new a hypothesis.

Introduction
Chronic subdural hematoma (CSH) is an abnormal collection of liquid blood between the dura mater and the arachnoid mater which is frequently encountered in neurosurgical conditions, especially in the elderly (1,2). Various ethological factors have been identified but head trauma is the most common (3). Surgeons may encounter unwanted postoperative complications such as cerebral edema, tension pneumocephalus, recurrent hematoma, seizure, failure of the brain to re-expand (3-5). But intraventricular hemorrhage (IVH) after evacuation of CSH is a rarely encountered complication. We report a patient who had an isolated IVH which was identified following evacuation of left frontotemporalsubaparietal CSH.
Case Report

A 72-year-old female patient was referred to the emergency room with right hemiparesis and two days of complaints of confusion. Right hemiparesis, lack of orientation and pathological reflexes were identified in her first neurological examination. Also, we learned from her children that she had suffered a head trauma three weeks previously and she did not take an anticoagulant therapy. Cranial Computed Tomography (CT) and Cranial Magnetic Resonance Imaging (MRI) were performed, and chronic-subacute subdural hemorrhage and septations with 1.2 centimeter midline shift were identified on CT (Figure 1A) and on MRI (Figure 1B). We decided to perform urgent surgery via burr hole exploration. Informed patient consent was obtained from her guardians. The patient’s body and head were laid on her right side and then burr hole was made in the left frontal bone with a high speed drill under local anesthesia and intravenous sedation. The external membrane of the chronic subdural hemorrhage at the left frontal bone was coagulated with bipolar and then opened. Compressive hemorrhage in the subdural space leaked out on its own. Frontal feeding catheter was placed in the subdural space. The subdural space was washed with saline. The frontal part of the internal membrane of the CSH was opened under the operating microscope with a bipolar. The frontal burr hole feeding catheter was left in place. The feeding catheter was connected to a closed drainage system. A cranial CT was performed on the 3rd postoperative day (Figure 2A) and then the feeding catheter was withdrawn. Bilateral IVH was observed in the occipital horns of the lateral ventricular, but there was no midline shift. The second cranial CT was performed on the 6th day of postoperative (Figure 2B). We observed hemorrhage in the occipital horn of the lateral ventricular again, but there was no acute hydrocephalus. The patient was discharged on the 7th day after surgery with complete recovery.

Discussion

Different bleeding patterns have been defined after evacuation of subdural hemorrhage in the literature. Cortical hyperemia beneath the hematoma, subarachnoid hemorrhage (SAH), supratentorial intracerebral, intraventricular, and retro cerebellar hemorrhages are rarely reported (6). In a large series of 1000 cases CSH, 4% cases of post-operative intracranial bleeding were described (7). Rusconi et al, have been reported unusual post-operative hemorrhagic events with an incidence of 0.78% (3 patients) (6). Other authors have been reported an incidence range between 0.2-4% (8,9). Overdrainage, rapid brain decompression and shift of the intracranial contents, massive cerebrospinal fluid loss, venous outflow impairment and vascular dysregulation with blood flow increase, are the mechanisms currently debated (6).

Two possible mechanisms have been suggested to explain isolated IVH after evacuation of CSH. The first of these was suggested by Cook and colleagues. Their hypothesis was that elderly patients with physiological

Figure 1. Preoperative axial cranial CT: There was no hemorrhage in the lateral ventricular before surgery (Fig 1A), Preoperative T2 weighted axial cranial MRI: Left frontotemporoparietal subacute-chronic subdural hemorrhage with 1.2 centimeters midline shift (Fig 1B).

Figure 2. First axial Cranial CT after surgery: Midline shift recovered but a new intraventricular hemorrhage was detected (White arrow) (Fig 2A), Second axial Cranial CT after surgery: neither increase in intraventricular hemorrhage nor development of hydrocephalus were detected (Fig 2B).
Aging of the cerebral vascular tree may not tolerate a sudden restoration of normal perfusion pressure in areas of deranged cerebral vascular autoregulation (5,10). The second hypothesis was suggested by Savadekar and Salunke. They hypothesized that after sudden decompression of the brain, the differential expansile qualities of solid (brain) and liquid (CSF) components of the cranium may result in mechanical stress at the interface. It may then cause rupture of the engorged subependymal veins (2). In addition to these, Muneza and colleagues suggested that fragile cerebral vessels, direct vascular damage and a shift of the midline structures might be contributory factors for IVH (4).

Our case was similar to the literature. The patient was elderly, and she had a subacute-chronic subdural hematoma which included a 1.2 centimeter midline shift. CSH was evacuated via burr holes under local anesthesia and intravenous sedation. We identified an isolated hemorrhage in the lateral ventricular occipital horns on the 3rd day after surgery. Our opinion is that when evacuation of CSH was started suddenly it might cause rapid correction of midline shift. Rapid correction of the midline shift and rapid movement of the brain might lead to vascular shearing owing to mechanical stress especially on choroid plexus vessels. The elderly may be susceptible to this vascular shear due to vascular aging. Our hypothesis seems to be supported by two hypotheses. In addition to these, the other causes of isolated IVH include coagulation disorders, pituitary apoplexy, sickle cell anemia, drug consumption and vasculitides, but none of these was demonstrated. In our opinion is that, isolated IVH is a different event and may not be explained with vascular self-adjustment which was suggested to explain for intracerebral hemorrhage and SAH following removal of the subdural hemorrhage. Multil surgical techniques have been defined in the literature for evacuation of chronic subdural hemorrhage, including twist drill craniostomy, burr-hole craniostomy, and craniotomy. And various complication have been reported related with surgical techniques (11). In our case, we have prefered the burr hole craniostomy for evacuation of subacute-chronic subdural hemorrhage under the local anesthesia and intravenous sedation. Our opinion is that gradual and graded evacuation can be archived by covering of the burr hole with a sponge or a cotonoid immedietly after opening the dura and the outer membrane. And than, A sponge or a cotonoid can be removed intermittent. By that mechanism, evacuation of subdural hemorrhage can be controlled and slowed down in a controlled way and this procedure can be continued till the subdural pressures equalize with the atmospheric pressures. This gradual and graded evacuation of subdural hemorrhage may decrease the mechanical stres on choroid plexus vessels.

In conclusion, although isolated IVH is rarely encountered, surgeons should keep it in mind because IVH may result in lethal complications following evacuation of CSH. Clinical awareness of this complication is essential. We suggest that slow and gradual decompression may protect the patient from this complication. Aging, amount of midline shift and speed of correction of midline shift may be contributory factors. Causative factors have not yet been clearly identified. Therefore, further investigations and large patient series are needed to clarify this phenomenon.

References
8. Dinc C, Iplikcioglu AC, Biktiaz K, Navruz Y. Intracerebellar

