CASE REPORT

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Epidural Hematomas due to Occipital Artery Injury Following Ventriculoperitoneal Shunt and Extraventricular Drainage

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Ventriculoperitoneal (VP) shunt is a common treatment for hydrocephalic patients. However, complications, such as shunt tube occlusion, infection, intracranial hemorrhage, seizure can occur. Of these, intracranial hemorrhage may occur due to intracranial vascular injury or a rapid decrease of intracranial pressure (ICP). Most of these hemorrhages are subdural hematomas (SDH) while a few are epidural hematomas (EDH). It is extremely rare for an intracranial hemorrhage to occur due to an extension of the bleeding from an injured extracranial vessel. We report two cases of EDH due to occipital artery injury following VP shunt and extraventricular drainage (EVD).

KEY WORDS : Epidural hematoma · Ventriculoperitoneal shunt · Extraventricular drainage · Occipital artery.

Introduction

Ventriculoperitoneal (VP) shunt is a common option for hydrocephalic patients in neurosurgery, but it may be associated with many complications such as shunt tube occlusion, infection, intracranial hemorrhage, and seizure[4,5,7]. Of these complications, intracranial hemorrhage occurs most commonly due to intracranial vascular injury or a rapid intracranial pressure (ICP) decrease caused by the overdrainage of cerebrospinal fluid (CSF)[1,3,5,7]. Most of intracranial hemorrhages resulting from low ICP are subdural hematoma (SDH). Epidural hematoma (EDH) may also develop, but it is uncommon and reported only occasionally[1-3,5,7].

Bleeding from an extracranial vascular injury can extend into the intracranial space along the shunt tube. It can lead to intracranial hemorrhage, but it is very rare. The authors report two cases of EDH due to occipital artery injury following VP shunt and extraventricular drainange (EVD) with a review of the literature.

Case Report

Case 1

A 66-year-old male patient underwent surgery for a left anterior communicating artery aneurysm rupture in April 1993. He later had a decompressive craniectomy for traumatic SDH in September 2003, and a cranioplasty in December 2003. He had been well until he

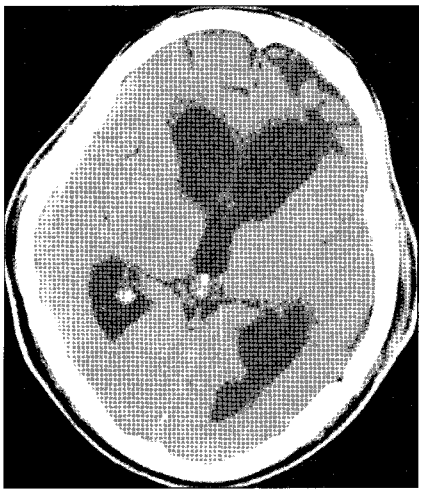

Fig. 1. Preoperative initial computed tomography (CT) showing residual cerebromalacia in left frontal lobe and diffuse both lateral ventricles enlargement.

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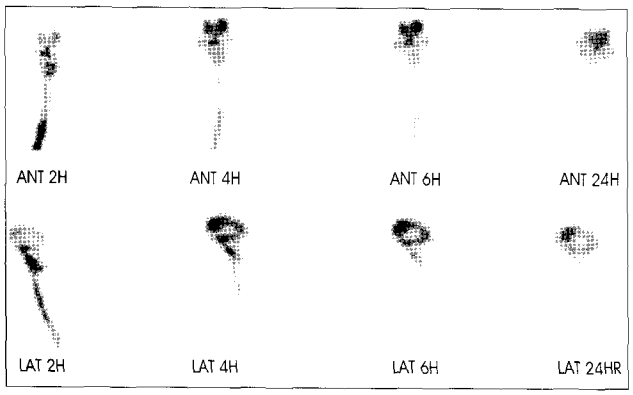

Fig. 2. Preoperative radioactive isotope cerebrospinal fluid (CSF) flow study showing delayed CSF migration with ventricular reflux and dilatation of lateral ventricles.

was readmitted to our department due to ambulatory difficulty and memory disturbance in February 2004. A brain computed tomography (CT) revealed diffuse enlargement of both lateral and third ventricles (Fig. 1). On a radioactive isotope CSF flow study, he was diagnosed with a communicating hydrocephalus (Fig. 2) and underwent a VP shunt operation.

After the operation was performed, he gradually became stuporous state. An emergent brain CT showed a small amount of intracerebral hemorrhage (ICH), intraventricular hemorrhage (IVH), and subcutaneous hematoma (Fig. 3). After two and a half hours, his consciousness fell into a deep stupor, the right pupillary reflex disappeared, and subcutaneous swelling

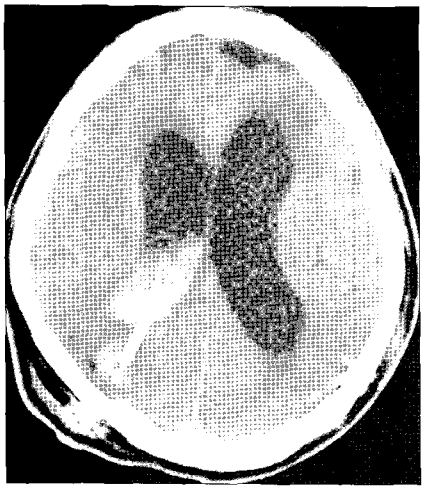

Fig. 3. The first postoperative brain CT showing the development of the focal intracerebral hemorrhage in the right parietal lobe, intraventricular hemorrhage and subcutaneous hematoma in the posterior nuchal scalp following ventriculopetitoneal shunt tract.

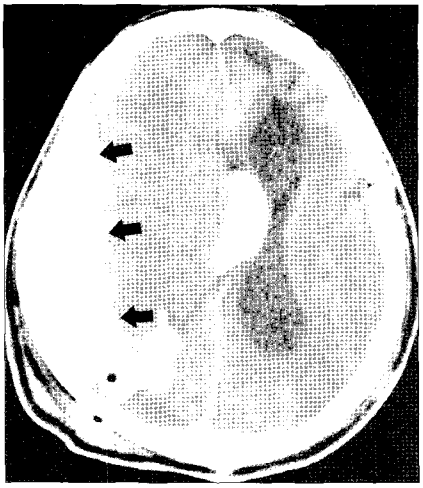

Fig. 4. The second postoperative brain CT showing the acute epidural hemorrhage in the right frontotemporoparietal area with subfalcial herniation and an increased amount of hemorrhage along the shunt tube.

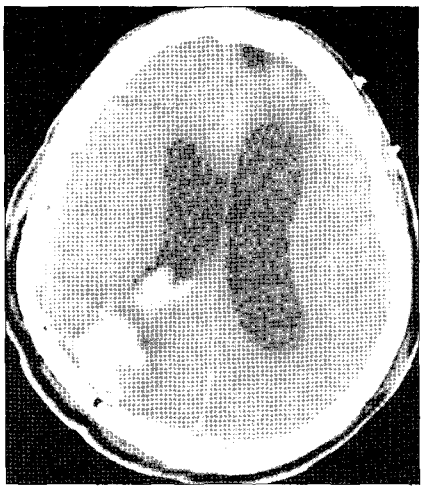

Fig. 5. The second postoperative brain CT showing the removed acute epidural hemorrhage and a slightly decreased amount of hemorrhage along the shunt tube, and improvement of the subfalcial herniation.

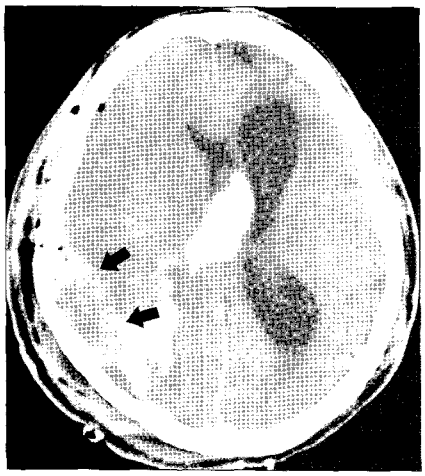

Fig. 6. The second postoperative second brain CT showing the recurrence of the acute epidural hemorrhage in the right parietooccipital area and an increased amount of hemorrhage along the shunt tube. The subfalcial herniation is showing again due to this rebleeding.

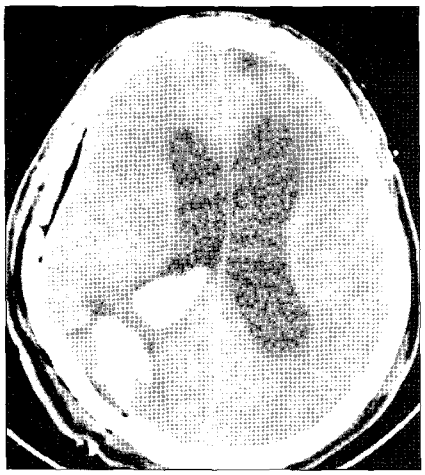

Fig. 7. The third postoperative brain CT showing the removal of acute epidural hemorrhage and a slightly decreased amount of hemorrhage along the shunt tube, and improvement of the subfalcial herniation.

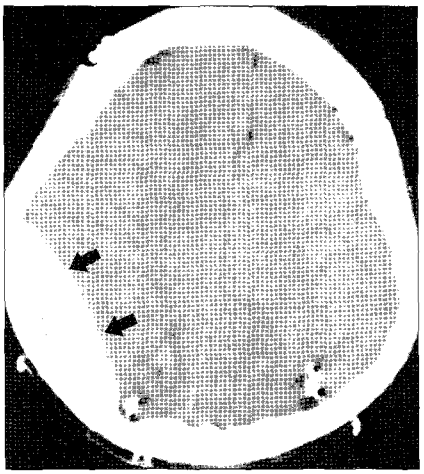

Fig. 8. Postoperative brain CT showing the development of the acute epidural hemorrhage in the right parietoocipital area beside the extraventricular drainage (EVD) tube.

was worsened. A repeated brain CT showed a large amount of EDH around the burr hole which was compressing the right lateral ventricle and brainstem (Fig. 4). An emergency decompressive craniectomy and evacuation of hematoma was performed, but the bleeding focus was not evident. After the surgery, his consciousness was still in a deep stupor but the right pupillary reflex showed improvement. A postoperative brain CT showed decreased amount of the EDH and remarkable improvement in the right ventricle and brainstem compression, but a small amount of ICH and IVH were still remained (Fig. 5).

After ten hours, his right pupillary reflex disappeared again and subcutaneous swelling developed repeatedly. On a repeated brain CT, the EDH, ICH and IVH recurred and the right lateral ventricle and brainstem were compressed again, and the subcutaneous hematoma under the scalp also appeared (Fig. 6). The second emergency operation was performed. However, we could not find the bleeding focus in the intracranial space. Instead, there was a large amount of bleeding in the occipital subcutaneous area. We extended the incision of the scalp downward along the shunt tube's path and closely observed the path until we found bleeding from the injured occipital artery. After exposing the bleeding focus, we coagulated the injured occipital artery and sutured the operation field. His consciousness was still deep stupor, but the right pupillary reflex became improved to prompt. A postoperative brain CT did not show anymore EDH (Fig. 7). A few months later his conscious level had improved to deep drowsy, both pupillary reflexes were intact, and he was discharged in a bedridden state.

Case 2

We experienced another case in an 11-year-old boy who was diagnosed with craniopharyngioma after brain imaging study.

The hydrocephalus developed in this patient due to a partial obstruction of the third ventricle by a tumor. After craniotomy and tumor removal, the ventricle continued to become enlarged. His consciousness gradually deteriorated from deep drowsy to stuporous, so an EVD was performed at the both parietooccipital points. On post-EVD brain CT, there was scalp swelling with subcutaneous hematoma and EDH around the right burr hole (Fig. 8).

Immediately after the scalp revision, we found continuous bleeding from the injured occipital artery. After the bleeding was controlled, the EDH did not expand any further and consciousness improved to drowsy. A few days later, the EVD was removed and the hydrocephalus recurred. His parents gave up further treatment, and he became comaous and expired.

Discussion

VP shunt is known to be associated with more complications than any other neurosurgical operation. Complications following VP shunt are shunt tube occlusion, infection, intracranial hemorrhage, overdrainage of CSF, seizures, etc. It has been reported that 50% of patients who undergo VP shunt operation have complications[4-7]. Among these complications, intracranial hemorrhage usually results from low ICP due to overdrainage of CSF. The hemorrhage is usually SDH, and rarely EDH. It occurs in about 0.4% cases of VP shunt and EVD[1-3,5,7]. The mechanism of EDH after VP shunt and EVD is not exactly known. Most authors have postulated that a large amount of CSF drainage leads to a rapid decreasing of ICP, seperating dura matter from the skull and makes an epidural space. When the small veins of the dura are torn in this space, the bleeding is thought to produce EDH[1-3,5,7]. In children, the adhesion of dura matter to skull is weaker than that in adults. In those with trauma or infection history, the adhesion of dura matter to the arachnoid membrane is stronger, and therefore EDH can develop more easily[1-3,5,7]. Because the volume of brain parenchyme in hydrocephalus patients is less than that of a normal person, a larger EDH can occur[5,7]. Reported cases of EDH produced through this mechanism are rare, but Weiss et al.[8] reported one such case. Reports of the EDH originating from extracranial vascular injury are extremely rare, and are very difficult to predict, especially when the bleeding extends through the intracranial space along the shunt tube. EDH produced in this manner has a high risk of expansion as the ICP decreases due to further CSF drainage. We believe this may cause further brain damage.

Consequently, it is not only important to pay attention to prevent the rapid decrease of ICP, but we must also keep in mind that intracranial hemorrhage can occur by extracranial vascular injury. Hence, operators need to pay close attention to extracranial vascular injury and appropriate treatment during VP shunt or EVD.

Conclusion

We experienced two cases of EDH produced from extracranial space, and not from the intracranial origin. These were produced by the extension of the bleeding from the occipital artery injury. Accordingly, operators should pay a special attention and provide meticulous care to extracranial vascular injury and appropriate treatment during VP shunt or EVD.

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