

Sinking Skin Flap Syndrome after Craniectomy in a Patient Who Previously Underwent Ventriculoperitoneal Shunt

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Sinking skin flap syndrome, resulting from decompressive craniectomy, is defined as a series of neurologic symptoms with skin depression at the site of cranial defect. A 61-year-old male was hospitalized with high fever and operative site swelling. He underwent decompressive craniectomy on his left side for treatment for acute subdural hematoma and traumatic intracerebral hematoma 5 years ago. Four months later, a ventriculoperitoneal shunt was performed for treatment for hydrocephalus and cranioplasty was also performed. We suspected infection at the previous operative site and proceeded with craniectomy and epidural abscess removal. Following the procedure, the depression of the sinking flap became significant, and he has suffered from right hemiparesis. We performed a shunt catheter tie at the level of the right clavicle under local anesthesia, and the patient recovered his health to his baseline. We present a patient who was successfully managed with a tie of the shunt catheter for sinking skin flap syndrome.

(Korean J Neurotrauma 2012;8:149-152)

KEY WORDS: Decompressive craniectomy · Sinking skin flap syndrome · Ventriculoperitoneal shunt.

Introduction

Sinking skin flap syndrome (SSFS) is defined as serious disabling neurologic deficits and impairment of general status with concave deformity and relaxation of the skin flap and it tends to develop several weeks to months after large craniectomy.^{1,5)} The pathophysiology include cerebrospinal fluid (CSF) hypovolemia and development of an atmospheric pressure gradient that may be aggravated by CSF diversion, dehydration, and position change.^{7,10)} The neurologic symptoms of SSFS include headache, vertigo, tinnitus, fatigue, loss of concentration, loss of memory, depression, dysphagia, apraxia, paresis of extremities, and convulsion.^{2,5)} SSFS that occurred after ventriculoperitoneal (V-P) shunt for craniectomy overcame by temporarily increasing the pressure of a programmable pressure valve to allow for the expansion of a depressed scalp flap and facilitate subsequent

cranioplasty.^{4,9)}

However, we needed to place a fixed type V-P shunt valve to our patient, so we performed a tie of the shunt catheter to block CSF drainage. This paper reports an experience of treating SSFS by tying the shunt catheter in a patient who underwent placement of a fixed type V-P shunt valve.

Case Report

A 61-year-old male was transferred to our hospital with stuporous mentality developed by complications from a fall 5 years ago. Initial brain computed tomography (CT) scans showed acute subdural hematoma and traumatic intracerebral hematoma on the left frontotemporal region with significant mass effect and midline shift to the right (Figure 1A). An emergency decompressive craniectomy and hematoma evacuation were performed to prevent the further complication. The postoperative CT scan showed resolution of the subdural hematoma and restoration of the significant midline shift. The patient gradually recovered ability to follow simple commands, such as hand grasping and leg elevation. However, 4 months later, the CT scan showed enlargement of the ventricular system, and the patient suffered

Received: May 29, 2012 / **Revised:** July 16, 2012

Accepted: July 16, 2012

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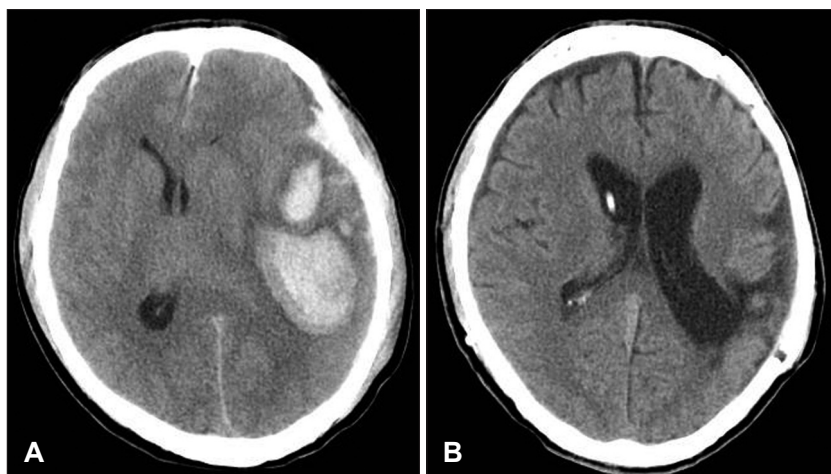


FIGURE 1. Brain computed tomography (CT) showed acute subdural hematoma and traumatic intracerebral hematoma on the left frontotemporal region with significant mass effect and midline shift to the right at 5 years ago (A) and brain CT after ventriculoperitoneal shunt via right Kocher's point and cranioplasty were performed (B).



FIGURE 2. Brain computed tomography after 5 years. It revealed a low density around the left frontotemporal bone and soft tissue swelling.



FIGURE 3. Brain computed tomography after bone flap removal. It revealed marked concavity of the brain at the defect site of the craniectomy associated with severe midline shift to the right.

from urinary incontinence. A V-P shunt via right Kocher's point with MEDTRONIC Strata[®] valve at 2.0 setting and cranioplasty were performed simultaneously (Figure 1B). After surgery, the patient was discharged after the gradual improvement of his mentation.

Five years later, he returned to the hospital, present with high fever and operative site scalp swelling. After the neurologic examination, including grade V motor power, he was within the normal limit. However, the CT scan showed a low density around the left frontotemporal bone and mild midline shift (Figure 2). In conclusion, we suspected infection of the previous operative site, so we have performed craniectomy and epidural abscess removal. *Klebsiella pneumoniae* was cultured from the surgical site, and antibiotics were given intravenously for two weeks. However, two days after surgery, the patient suffered from right hemiparesis, motor grade II. The follow-up CT scans revealed marked concavity of the brain at the defect site of the craniectomy associated with midline shift to the right (Figure 3). First, we used

adequate hydration and placed the patient in Trendelenburg position. It was not effective, but we immediately performed a tie of the shunt catheter at the level of the right clavicle under local anesthesia. Within 12 hours, the motor grade of the patient has improved. The follow-up CT scan showed restoration of the midline shift without significant complication compared to the previous image (Figure 4).

Discussion

Decompressive craniectomy is most commonly used in the neurosurgical field for the treatment of intractable intracranial hypertension in patients with head injury, acute stroke, and severe brain edema after an intracranial procedure.¹⁰⁾ The value of this surgical procedure in achieving a better outcome is controversial.^{12,14)} Typically, a decompressive craniectomy may reduce intracranial pressure by producing more space for the edematous brain tissue when a portion of the skull is removed. However, many complications can follow



FIGURE 4. Follow up computed tomography scan shows restoration of the midline shift without significant complication compared to the previous image.

a decompressive craniectomy and have adverse effects on the patient's neurological recovery. Among many adverse effects, SSFS is one of them.^{2,13)}

SSFS was first reported in 1997 by Yamamura et al.¹⁵⁾ SSFS is defined as a series of neurologic symptoms with skin depression at the site of cranial defect; it develops several weeks to months after large external cerebral decompression. Yamamura et al.¹⁵⁾ reported that some patients with a depressed skin flap after external cerebral decompression showed improved neurologic symptoms after cranioplasty to correct tissue formation under the flap. Thus, SSFS is characterized by rapid improvement of neurologic symptoms after cranioplasty.

Many authors have attempted to explain the pathophysiology of this phenomenon. One theorized that atmospheric pressure is directly transmitted to the intracranial cavity, causing inward shifting of the scalp over the craniectomy site.⁸⁾ According to this theory, George et al.³⁾ showed in a series of angiographies that there was a correlation between restoration of the midline shift and clinical improvement following cranioplasty. Recently, several authors^{11,13)} proposed that negative gradients between atmospheric and intracranial pressure, which is aggravated by changes in the CSF compartment following CSF hypovolemia, to be the mechanism of neurological deterioration after craniectomy.

Post-traumatic hydrocephalus (PTH) is common in patients who have undergone a decompressive craniectomy after severe traumatic brain injury and can occur among 10% to 40% of these patients.^{1,6)} The CSF drainage in patients suffering from hydrocephalus exacerbates this effect by creating a pressure gradient through the craniectomy site. After V-P shunt, this effect may aggravate and cause neurological deterioration such as that found in SSFS.

Thus, the goal of treating SSFS is restoration of the pressure exerted by depression of craniectomy site. Han et al.⁴⁾ suggested that SSFS overcome in a patient by temporarily increasing the shunt by adjusting the shunt pressure using a programmable V-P shunt valve to allow for expansion of a depressed scalp flap and facilitate the subsequent cranioplasty. Adjusting the shunt pressure may facilitate dissection during the subsequent cranioplasty and decreases the incidence of intracranial infection, hematoma, and effusion by eliminating the dead space.⁹⁾

We have used a pressure type valve, i.e., fixed shunt valve; the pressure cannot be adjusted in this type of valve. The shunt catheter tie uses the same technique as adjusting shunt pressure with a valve. We hypothesized that the obstruction of the CSF pathway via a tie of the shunt catheter has the same effect on increasing pressure as temporarily increasing the pressure of a programmable valve shunt. First, we palpated the shunt catheter at the level of the right clavicle and vertically incised the skin about 1 cm and dissected subcutaneous tissue in order to find the shunt catheter. We placed the shunt catheter into a 'C' shape, and the proximal end of the 'C' shunt catheter was tied with 5-0 silk. If the patient later suffers from hydrocephalus, we will be able to easily find and stitch out the tie site of the shunt catheter. This procedure is simple and can be done in a general ward. Also, this procedure cause less damage toward patients, including those that are medically compromised, than shunt catheter removal under general anesthesia. The patient had good results without any complications. However, the patient may need cranioplasty resulting from another incidence of SSFS in the future. In conclusion, a tie of the shunt catheter proved to be safe and effective in the case presented here.

Conclusion

Our case report includes a patient with sinking skin flap syndrome who was treated with a tie of the shunt catheter without complications. We suggest that the safe and effective surgical method is to expand the scalp depression and eliminate the dead space in the V-P shunt with temporary occlusion of the shunt by tying the shunt catheter before cranioplasty. Further clinical study will be needed to investigate SSFS.

■ The authors have no financial conflicts of interest.

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