

Sinking Skin Flap Syndrome after Hemicraniectomy and Ventriculo-Peritoneal Shunt Overdrainage

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Abstract

We present a case of sinking skin flap syndrome after hemicraniectomy and insertion of a ventriculo-peritoneal shunt following severe craniocerebral injury. When the neurological status deteriorated due to cerebrospinal fluid overdrainage and resulting midline shift, complete ligation of the vp shunt became a temporary solution before definitive cranioplasty could be performed.

Keywords: Sinking skin flap syndrome; vp shunt; Shunt overdrainage; Craniectomy; Cranioplasty

Introduction

The sinking skin flap syndrome is a rare complication after hemicraniectomy and is characterized by the occurrence of headache, epileptic seizures, vertigo, dysesthesia, palsy and reduced vigilance [1,2]. It is considered to be caused at the site of the defect by atmospheric pressure, reduced cerebral blood flow (CBF) and altered cerebrospinal fluid (CSF)-pressure – or a combination thereof [1]. Atmospheric pressure may lead to direct cortical compression [2]. Improvement of CBF at the site of the craniectomy and the opposite hemisphere following cranioplasty has been shown by radiological studies including xenon computertomography (CT), perfusion CT, and dynamic CT [3-5]. A decrease in CSF pressure, due to changes in posture, but also caused by overdrainage after shunt insertion, may result in collapse of brain structure [6,7,1].

Case report

A 46-year-old man suffered a brain injury with left-sided subdural and epidural hematoma, requiring left-sided decompressive hemicraniectomy. In addition, a ventriculo-peritoneal (vp) shunt was inserted because of posttraumatic obstructive hydrocephalus using a valve-opening pressure (vop) of 130 mmH₂O. Clinically, the patient showed a residual hemiparesis and right-sided hemispasticity. Three months after the accident cranioplasty was performed. Two years later, the cranioplasty dislocated with a skull depression of 2 cm and had to be removed. After refixation, an intracranial large hygroma developed and consequently the vop was increased to 200 mmH₂O. Subsequently, an epidural abscess developed and the cranioplasty had to be removed. One week later, the patient presented with a fluctuating level of consciousness and was admitted to our hospital.

On examination, the patient was somnolent and showed a marked right-sided hemiparesis and tetraspasticity. On admission, a CT scan revealed a slit left lateral ventricle and compression of the right lateral ventricle due to midline shift (Figure 1). We suspected overdrainage of the vp shunt and after interdisciplinary discussion complete surgical ligation of the vp shunt was performed leading to major clinical improvement: the patient was awake and was able to interact with his environment using single words and head movements. A follow-up CT scan showed ventricular expansion on the left and reduced midline shift (Figure 2). The patient could be discharged and completed his rehabilitation treatment, before he underwent cranioplasty 6 weeks later (Figure 3). The timepoint for cranioplasty was chosen in regard to the previous epidural abscess and the clinical condition of the patient. During this procedure the ligation was removed and the vop was again set at 200 mm H₂O.



Figure 1: CT Scan on admission

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Figure 2: CT Scan 2 days after shunt ligation



Figure 3: CT Scan after cranioplasty

Discussion

Vp shunt overdrainage and thus compression of the brain by atmospheric pressure following large hemicraniectomy led to

neurological deterioration in our patient. This phenomenon is known as sinking skin flap syndrome (SSFS). The pathogenesis of SSFS in our patient is supported by the observation that only complete surgical ligation of the vp shunt reverted the midline shift and normalized the patient's level of consciousness. The evidence-based treatment of this syndrome is prompt cranioplasty [8]. In our case, this procedure could not be performed because of the previous infection. Therefore, ligation of the shunt was performed as a temporary solution and resulted in clinical improvement. Slow reduction of the vop is further planned in order to decrease the ventricle size.

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