Epidural Blood Patch for Severe Postoperative Intracranial Hypotension

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Abstract: Brain sag is a rare but serious postoperative complication after craniotomy. It is a clinical entity that refers to severe cerebrospinal fluid hypovolemia causing acute neurologic decompensation and obtundation. The established treatment is trendelenberg positioning. We present 3 cases of severe brain sag in which patients developed acute neurologic deterioration unresponsive to conventional treatment. An epidural blood patch was performed emergently to prevent further neurologic decline. The administration of epidural blood patch resulted in immediate and dramatic reversal of obtundation in each of these patients. Epidural blood patch may be a life-saving intervention in postcraniotomy patients presenting with refractory brain sag. It should be offered to patients who meet the criteria for brain sag and are unresponsive to conventional treatment modalities, despite the clinical presentation of herniation and coma.

Key Words: epidural blood patch, brain sag, postoperative intracranial hypotension

Brain sag is a serious postoperative complication defined by transtentorial brain herniation as a consequence of severe intracranial cerebrospinal fluid (CSF) hypovolemia after craniotomy. It presents as acute neurologic deterioration with imaging studies showing empty ventricles and a downward shift of the cerebellar tonsils. Typically, brain sag develops between 1 and 4 days postoperatively and the hallmark clinical signs are anisocoria with acute mental status decline. Typically the symptoms of brain sag are reversible with Trendelenberg positioning and the only established standard treatment is Trendelenberg positioning.1

Epidural blood patch is the definitive treatment of postdural puncture headache resulting from diagnostic lumbar puncture, spinal anesthesia, inadvertent dural puncture during attempted epidural space entry, or intrathecal chemotherapy.2 There are also numerous reports that describe epidural blood patch (EBP) in treatment of postural headaches resulting from spontaneous CSF leaks and primary intracranial hypotension.

This manuscript describes 3 cases of severe obtundation from brain sag, which were refractory to conventional treatment with trendelenberg positioning. EBP was used to reverse these symptoms successfully. EBP is not yet a therapy of proven efficacy for the routine treatment of brain sag. However, on the basis of these 3 cases it may be considered in cases that are refractory to conventional treatment.

CASE 1

A 36-year-old woman with a grade 4 subarachnoid hemorrhage underwent right fronto-temporal craniotomy for choroidal aneurysm clipping. A lumbar spinal drain was placed intraoperatively to facilitate brain retraction. The intraoperative course was uneventful and she was extubated immediately postoperatively after removal of lumbar spinal drain. On postoperative day 1, she deteriorated acutely developing mental obtundation and anisocoria and required emergent reintubation. Intracranial pressure was 0 mm Hg and Glasgow Coma Scale (GCS) was 5. Vasospasm was ruled out by angiography. Brain computed tomography (CT) revealed right frontal subdural fluid, empty ventricles, and basilar cistern effacement. Brain sag was diagnosed based on the presentation of acute mental status decline, anisocoria, and severe intracranial CSF hypovolemia evidenced by the presence of empty ventricles and effacement of basilar cisterns (Fig. 1). In the setting of severe brain sag presenting with extensor posturing that was unresponsive to Trendelenberg positioning, an emergency EBP was considered. A lumbar EBP using 20 mL of autologous blood was performed at the bedside in the Neurological ICU (NICU) at the L3-L4 interspace. Immediately after completion of the procedure the intracranial pressure rose to 5 mm Hg. Dramatic improvement of mental status was noted in the next 2 hours. She was extubated 12 hours later with a GCS 13 and no focal neurologic deficits. She remained clinically stable and was discharged home 2 weeks later.

CASE 2

A 26-year-old man underwent craniotomy for left ophthalmic artery aneurysm clipping. A spinal drain was placed intraoperatively and CSF was drained intermittently during the procedure at surgical discretion. He had an uneventful intraoperative course and after removal of the lumbar drain he was extubated and taken to the NICU.
On the first postoperative day, he developed third nerve paresis, severe headache with waning mental status. CT scan revealed pneumocephalus and subdural hematoma for which he underwent evacuation of epidural air and subgaleal drain placement. However, he continued to have a severe postural headache, worsening lethargy, and anisocoria over the next 3 days. Brain CT revealed right shift, 1 cm subfalcine herniation and effacement of perimesencephalic cisterns.

The criteria of brain sag was met by the presentation of third nerve paresis, acute mental status decline with imaging studies that reflected the CSF hypovolemia on the basis of the presence of empty perimesencephalic cisterns.

An EBP was performed using 15 mL of autologus blood. The end point of autologous blood injection was pressure in the lower back reported by the patient. He had immediate resolution of headache with improvement of mental status. Brain CT 12 hours later showed full ventricles and basilar cisterns. He was discharged 4 days later in good condition.

### CASE 3

A 62-year-old woman presented to the NICU after becoming suddenly unresponsive at home requiring cardiopulmonary resuscitation and intubation. She underwent an emergent craniotomy for grade 4, Fisher III subarachnoid hemorrhage. At the end of surgery the lumbar spinal drain was removed but she was kept intubated and transferred back to the NICU.

After extubation on the first postoperative day, her mental status progressively deteriorated. She became unresponsive and required emergent reintubation for airway protection on operative day 3. After ruling out vasospasm by angiography, brain CT scan showed cerebral edema, empty ventricles, effaced basilar cisterns, crowding of posterior fossa elements, and transtentorial herniation.

In this case brain sag was diagnosed based on the acute worsening of mental status worsening leading to obtundation and the characteristic appearance of intracranial CSF hypovolemia as evidenced on CT scan by empty ventricles and effaced cisterns.

As a result of the patient’s rapid deterioration and lack of response to trendelenberg positioning, a lumbar epidural blood patch was done. This was done at the L5-S1 interspace with 10 mL of autologous blood. Blood (10 mL) was chosen empirically because the patient could not give feedback on side effects.

Twelve hours after the blood patch, the patient had reversal of mental status and was extubated. She was discharged in stable condition except for mild ataxia and short-term memory loss 7 days later after the blood patch.

### DISCUSSION

Brain sag has been identified as a relatively uncommon complication after craniotomy for brain aneurysm clipping. According to one study between April 2001 and June 2004 at Columbia University Medical Center, 16 aneurysmal subarachnoid hemorrhage patients were diagnosed with postoperative critical CSF hypovolemia, whereas 151 patients who underwent craniotomy for clipping were not.\(^3\)

The dural sac, CSF, brain, and blood constitute a closed system defined by the Monro-Kellie hypothesis.\(^4\) After craniotomy, the Monro-Kellie model no longer applies. Exposure to the atmosphere allows air entry into the subarachnoid space forming an air-fluid level. The third ventriculostomy, fenestration of the lamina terminalis, and the spinal drain results in CSF loss. CSF hypovolemia causes meningeal hyperemia, engorged venous sinuses, and pituitary hyperemia.\(^4\) CSF hypovolemia and subdural fluid collections makes the residual CSF critical to prevent caudal herniation and to keep the brain afloat.\(^1\)

Signs and symptoms of early tonsillar herniation are difficult to recognize in an unconscious patient. Changes in vital signs, mental status decline, pupillary asymmetry/ nonreactivity, and flexor/extensor posturing may be the only clinical clues.

Risk factors identified with brain sag include CT evidence of global cerebral edema at admission, prolonged operative time, ventriculostomy, and fenestration of the lamina terminalis and intraoperative lumbar drain.\(^1\)
The presence of preoperative global cerebral edema is a risk factor for development of postoperative brain sag because edematous brain parenchyma is more likely to experience downward traction and herniate with shifts in CSF dynamics. Longer surgery duration may reflect greater technical difficulty, allowing additional CSF loss during the case and thus stress postoperative CSF dynamics.³

Lumbar spinal drain placement influences the development of postcraniotomy critical CSF hypovolemia. Additionally, intraoperative fenestration of lamina terminalis and a third ventriculostomy serves as a significant conduit for CSF loss.³ Intraoperative spinal CSF drainage is frequently used during aneurysm surgery to reduce CSF volume and provide brain relaxation. Excessive CSF removal from the thecal sac clearly has the potential to cause downward traction on intracranial structures. Spinal drain placement directly violates the dura and creates a channel for continued CSF loss that may persist well after drain removal, thereby increasing the possibility of critical CSF hypovolemia during the postoperative period. Interestingly, spinal drain as a risk factor was not noted to be statistically significant as 94% of patients with brain sag had intraoperative spinal drainage versus 72% of control patients.³ Thus, brain sag is not a consistent consequence of lumbar spinal drain placement for reasons not yet fully understood.

The 3 criteria for diagnosis include imaging evidence of transtentorial herniation, effaced basal cisterns and oblong brainstem, and improvement of symptoms on Trendelenberg positioning.¹

The absence of a blood brain barrier in the pachymeninges causes a pressure gradient driven extravasation seen as pachymeningeal dural enhancement in contrast-enhanced imaging studies. Brain sag may not be easily evident on CT scan because axial views cannot see the pathology well. It is best seen on sagittal magnetic resonance imaging. Dural enhancement is seen early whereas subdural fluid collections and brain sag are indicators of an advanced state of pathology.⁵,⁶ Attempted drainage of subdural hematoma in patients with brain sag may cause worsening of symptoms.¹

Brain sag develops between 1 and 4 days postoperatively. The hallmark symptoms are anisocoria with acute mental status decline.¹ Compression of the ascending reticular activating system in the midline is responsible for change in mentation and obtundation.⁷ An increase in intracranial pressure after EBP probably provides the buoyancy to relieve compression of the midbrain and diencephalon thereby reversing the mental status changes in these cases.

Early recognition and treatment of this rare complication by increasing intracranial pressure and CSF volume is mandatory in treating these patients. Most of the patients who presented with brain sag at this institution have been successfully treated with Trendelenberg positioning. Patients who had recalcitrant symptoms had an EBP with dramatic reversal of neurologic status.

This postoperative syndrome has clinical features similar to those seen in spontaneous intracranial hypotension. The features of spontaneous intracranial hypotension may include brain magnetic resonance imaging that shows pachymeningeal enhancement, bilateral subdural collection, and downward displacement of the cerebellar tonsil.⁸ Many case reports have been described suggesting the use of EBP in the treatment of spontaneous intracranial hypotension. There is no consensus regarding the management of spontaneous intracranial hypotension. Despite the lack of an evidence base, treatments such as extended bed-rest, hydration, caffeine, and raising abdominal pressure have been described.⁹ If these are ineffective, lumbar epidural blood patch, if done early, seems to be effective.⁵ There is no definite or agreed-on standard approach in the literature. EBP seems to be associated with success: 2 series of spontaneous intracranial hypotension are reported in the anesthesiology literature.¹⁰,¹¹

The immediate effect of the epidural blood patch may be owing to tamponade of the thecal sac and an increase in CSF pressure with resultant reduction of the intracranial blood volume. In addition, epidural blood may cause an aseptic inflammation that seals the CSF leak and result in a vasoconstriction of epidural spinal vessels. In this limited series, we have not observed any relation between the amount of blood injected and the clinical outcome. Obtunded patients were empirically given between 10 and 20 mL of autologous blood. In an awake patient we used the end point as pressure or pain in the low back as reported by the patient as is our practice in blood patches for other indications.

CONCLUSIONS

In the setting of postcraniotomy brain sag resistant to other treatment, EBP may be a life saving procedure. As most patients with postcraniotomy brain sag respond to conservative treatment with Trendelenberg positioning, prophylactic blood patch after removal of the spinal drain is not indicated. Early recognition of brain sag is crucial and the administration of an EBP should not be delayed despite the presentation of transtentorial herniation and progressive obtundation.

Future studies are needed to ascertain the long-term sequelae, and optimal blood volume and level for epidural blood injection.

REFERENCES