**ABSTRACT**

Epidural hematoma is an indication for emergency neurosurgical intervention. This condition is an extremely rare postoperative complication of ventriculoperitoneal shunt and contralateral decompressive craniectomy. A 22-year-old male patient was admitted to our clinic with headache and a decline in the level of consciousness. We detected a left thalamic astrocytoma and hydrocephalus, which we treated via ventriculoperitoneal shunt surgery and ventricular drainage in emergency conditions. The patient experienced dysphasia on the first postoperative day and we found a right frontoparietal epidural hematoma. We evacuated the hematoma and exchanged the medium pressure valve for a high-pressure valve. The second patient was a 19-year-old male who had been assaulted. His pupils were fixed and dilated and had no reaction to painful stimulus. We detected bilateral frontotemporal skull fractures and right frontotemporoparietal subdural and epidural hematomas. We performed a right decompressive craniectomy and subdural/epidural hematoma evacuation followed by recovery under sedation in the intensive care unit. We performed cranial computed tomography six hours after surgery and found a left temporoparietal epidural hematoma. We performed a left temporoparietal craniotomy and epidural hematoma evacuation. The patient exhibited a higher level of consciousness and increased movement of his extremities. Epidural hematoma is a life-threatening complication encountered in neurosurgery practice. Neurosurgeons should be aware of the possibility of epidural hematoma following ventriculoperitoneal shunt or traumatic brain injury surgery.

**Keywords:** Epidural hematoma, Traumatic brain injury, Ventriculoperitoneal shunt

**INTRODUCTION**

Epidural hematoma (EDH) is a life-threatening indication for neurosurgical intervention. Ventriculoperitoneal shunt (VPS) is the most common procedure for the management of hydrocephalus. Subdural bleeding is frequently seen following VPS, but EDH is rare. Extra-axial mass lesions (subdural hematoma), extradural hematomas and intraparenchymal mass lesions (contusions and intracerebral hematomas) can be seen after traumatic brain injuries (TBI). Few cases of contralateral EDH following decompressive surgery for TBI have been reported. EDH is commonly seen in neurosurgical practice, but in this report, we present two extremely rare cases and causes of postoperative EDH.

**CASE REPORT**

**Case 1**

A 22-year-old male patient was admitted to the clinic with headache and a decline in the level of consciousness. We detected a left parietal astrocytoma and hydrocephalus (Figure 1 a-b). We first planned and performed right median pressure ventriculoperitoneal shunt surgery and left ventricular drainage in emergency...
conditions. We did not drainage cerebrospinal fluid (CSF) with ventricular drainage and it was for trapped CSF in the left occipital horn of lateral ventricle. Dysphasia and a decline in the level of consciousness appeared on the first postoperative day and we identified the presence of a left frontoparietal epidural hematoma (Figure 1c-d). We subsequently evacuated the hematoma via craniotomy and exchanged the medium pressure valve with one of high pressure. We discharged the patient as neurologically intact.

We performed left decompressive craniectomy and subdural/epidural hematoma evacuation. The patient was followed under sedation in the intensive care unit. Six hours after surgery we performed cranial computed tomography and found a right temporoparietal epidural hematoma (Figure 2d). We performed right temporoparietal craniotomy and epidural hematoma evacuation. The patient exhibited an increased level of consciousness and increased movement of his extremities.

**DISCUSSION**

Ventriculoperitoneal shunt surgery is the most common surgical procedure for hydrocephalus management. Following this surgery, intracranial hemorrhage sometimes arises because of ventriculostomy caused by overdrainage. Shunt obstruction and infection complications are more frequent than bleeding. Subdural hematomas are more common than EDH after ventriculostomy procedures. Cerebellar hemorrhages caused by cerebrospinal fluid decompression also are reported after VPS. Fukamachi et al. evaluated 1,055 postoperative cranial computed tomography images and determined that the EDH rate is 0.4% following VPS and ventricular drainage. Desai et al. evaluated 3,109 cranial operations and reported the rate for all intracranial postoperative hemorrhages as 1.9%.

The mechanism of EDH formation is not yet clear, but many authors suggest that falling intracranial pressure causes the cortex to collapse, tearing vessels attached to the dura. Seythanoglu et al. reported that skull-dura adhesions are more prominent than dura-arachnoid adhesions in some patients, thus bleeding occurs in the epidural space instead of the subdural space due to cerebrospinal fluid overdrainage. Other possible causes of hematoma include a coexistent bleeding disorder, bleeding from an occult dural vascular malformation and head trauma after surgery. In our first case, rapid intracranial pressure reduction with VPS could have led to detachment of the dura from the calvarium. We detected no vascular malformation during hematoma evacuation. The patient stayed in the intensive care unit and therefore had no trauma history. Hematological studies revealed no evidence of a bleeding disorder.

Sengupta and Hankinson reported three EDH complications following ventricular drainage among 22 patients and determined the underlying reasons as young age (<20 years old) and chronic hydrocephalus. Odake et al. discussed 43 supratentorial EDH cases after ventricular decompression in terms of cerebrospinal fluid drainage. They determined that the risk was higher in young and middle-aged patients, ages 10 to 40 years. Theoretically, the dura mater can be more easily detached from the calvarium in young patients. Fujimoto et al. reported prone surgery position as a further risk factor for EDH. Present patient’s single risk factor was his age. Age is important because dural-calvarial adhesions

**Figure 1:** (A) T1 axial weighted magnetic resonance images revealed a left thalamic astrocytoma; (B) hydrocephalus and trapped cerebrospinal fluid in the left occipital horn of lateral ventricle on computerized tomography; (C, D) left ventriculoperitoneal shunt catheter, right ventricle drainage catheter and also left temporoparietal epidural hematoma revealed on postoperative first day computerized tomography.

**Case 2**

A 19-year-old male was assaulted and admitted to the emergency unit with bilateral fixed, dilated pupils and unresponsiveness to painful stimulus. We detected bilateral frontotemporal skull fractures and left frontotemporoparietal subdural and epidural hematomas (Figure 2a-c).

**Figure 2:** (A) Left frontotemporoparietal subdural and epidural hematomas, midline shift and (B, C) bilateral frontotemporal skull fractures detected on computerized tomography and 3 dimensional reconstruction images. (D) Postoperative computerized tomography showed left sided craniectomy, right temporoparietal epidural hematoma and midline shift.
impairment, intraoperative fractures in injury after hematoma. et literature. cases intracranial condition. after ventricular endoscopic 44.2% mortality VPS. occurred drainage symptoms to hematoma. First, Byrappa if always bleed, patients can remain asymptomatic, delaying diagnosis and resulting in adverse outcomes. Byrappa et al. suggested two reasons for such a delay. First, VPS reduces intracranial pressure (ICP), while a hematoma causes a concomitant increase in ICP. Second, temporal and posterior fossa hematomas lead to rapid herniation, but frontoparietal convexity hematoma tends to spread throughout the entire hemisphere, delaying symptoms and diagnosis. Consequently, cases of chronic, calcified epidural hematomas after ventricular drainage have been reported.

EDH is a well-known complication of the VPS procedure. Louzada et al suggested using a programmable valve to prevent hematomas. However, Power et al and Harkness reported EDH cases that occurred following insertion of a programmable-valve VPS. In our first case, we evacuated the EDH with urgency and exchanged the medium-pressure valve for a high-pressure valve. Odake et al reported the mortality rate of EDH after ventricular drainage was 44.2% while 11.6% recovered with deficits. Although endoscopic third ventriculostomy also involves ventricular drainage, there are few EDH cases reported after endoscopic third ventriculostomy. Present patient was discharged neurologically intact.

Traumatic brain injury (TBI) is a life-threatening condition. TBI can cause subdural, epidural, and parenchymal hematomas and contusions with increasing intracranial pressure. EDH is extremely rare, with only 38 cases after decompressive craniectomy presented in the literature. Panourias et al, Matsuno et al, and Meguins et al reported contralateral epidural hematoma after decompressive craniectomy for adult subdural hematoma. Shen et al evaluated published data via Pubmed and found a rate of 2.4% for contralateral EDH after acute subdural hematoma. The average patient age was 35, males were in the majority and the main cause of injury was traffic accident. Unfavorable outcomes were reported for 70% of patients while 30% had favorable outcomes. The cases included 81% contralateral skull fractures and 76% intraoperative acute brain swelling. Our patient was male, 19 years old and had contralateral skull fractures.

Signs of a contralateral event after surgery include intraoperative brain swelling, postoperative neurological impairment, pupillary dilatation contralateral to the craniectomy side, grand mal seizure and intractable elevated ICP. Present patient had bilaterally dilated pupils preoperatively and brain swelling intraoperatively.

The pathophysiology of contralateral EDH after decompressive craniectomy is poorly understood, but possible underlying features include loss of tamponade effect, vasomotor mechanisms, and coagulopathy. The main cause appears to be corrupted equilibrium of the injured vessels and reactive ICP. Meguins et al recommends intracranial pressure monitoring of TBI patients, even if decompressive surgery is performed.

Possible sources of bleeding include a ruptured meningeal arterial branch, venous lacerations and skull fractures. Other possibilities include surgical decompression, CSF fistula, aggressive anti-edema treatment and systemic hypotension that induces intracranial hypotension. In present case, the patient had bilateral skull fractures, but there was no right epidural hematoma before the first operation. Thus, we believe that the mechanism in our case was loss of tamponade effect due to decompressive surgery and microvascular ruptures in the epidural space. In patients with intraoperative brain swelling or skull fractures, the development of contralateral hematomas should be considered after decompression of acute subdural hematoma.

CONCLUSION

Epidural hematoma has many causes so neurosurgeons should be aware of changes in neurological examination findings and intraoperative observations to avoid overlooking life-threatening surgical complications.

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