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Original Article

# Subdural effusion with ventriculomegaly after decompressive craniectomy for traumatic brain injury: A challenging entity

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ABSTRACT

*Problems considered:* Subdural effusion with concomitant ventriculomegaly (SEV) is not infrequently seen in patients with traumatic brain injury (TBI) after decompressive craniectomy (DC). The management of this entity remains very challenging. The aim of this study was to determine the incidence of SEV in patients who required DC.

*Methods:* This retrospective study was done over 8 months (March 2011–October 2011). All Patients with severe head injury (GCS ≤8) who developed subdural collection with associated ventriculomegaly in the postoperative period following DC were included in the study. Criteria for ventriculomegaly included modified frontal horn index greater than 0.3 and/or presence of periventricular lucencies.

*Results:* 270 patients underwent DC during the study period. SEV was seen in 80 (26.6%) patients. The mean age was 29.8 years with predominance of males (83%). Incidence of SEV was highest (59%) in the third week (day 14–21) after DC. 90% of SEV were ipsilateral to the side of craniectomy, and 81.25% ($n = 65$) were more than 10 mm in thickness. 47.5% ($n = 38$) of patients with SEV had IHH with mean thickness of 8.71 mm (range 3.5–23). 14 patients needed treatment in the form of various shunts. Of these the majority (86%) had concomitant IHH.

*Conclusions:* Contrary to common perception, SEV has a very high incidence (26.6% in our study) in patients who required DC following TBI. Our study shows that IHH is an important prognostic marker for the need of CSF diversion in these patients.



## 1. Introduction

Decompressive craniectomy (DC) is widely carried out in patients with raised intracranial pressure due to trauma or infarction. The procedure is not entirely free from complications. The incidence of subdural effusion after decompressive craniectomy is between 21 and 50%.[1] Subdural effusions are fluid collections which are usually asymptomatic, and the course follows spontaneous resolution. Nevertheless, they

*Abbreviations:* SEV, subdural effusion with concomitant ventriculomegaly; TBI, traumatic brain injury; DC, decompressive craniectomy; IHH, interhemispheric hygroma.

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occasionally become symptomatic due to associated ventriculomegaly and/or mass effect. Traumatic subdural effusion with ventriculomegaly (SEV) has been described in multiple case reports but its incidence and management has not been properly dealt with.[2–4] The term subdural effusion with ventriculomegaly (SEV) describes more accurately the nature and the severity of this condition, thereby differentiating it from the benign subdural collections of infancy and subdural hygromas.

External hydrocephalus is a well-established entity in infants which is benign and usually resolves without shunting.[5,6] The term "External Hydrocephalus" has also been used to describe the presence of extra ventricular cerebrospinal fluid (CSF) collections accompanied by hydrocephalus, particularly in cases of adults suffering from aneurysmal subarachnoid haemorrhage and severe head injuries.[4,7–10] However, the fact that this form of hydrocephalus may not have a benign course and needs in many cases surgical management demonstrates the need for a different term other than "external hydrocephalus."

The aim of this study was to determine the incidence of subdural effusion with ventriculomegaly in patients who required DC for traumatic brain injury and survived more than 3 days. A secondary objective was to study the role of interhemispheric hygroma (IHH) as a marker of impending hydrocephalus in these patients.

## 2. Material and methods

The retrospective study was conducted at Jai Prakash Narayan Apex Trauma Centre, All India Institute of Medical Sciences, New Delhi over a period of 8 months (March 2012–October 2012). All Patients with severe head injury (GCS $\leq$8) who developed subdural collection with associated ventriculomegaly in the postoperative period following DC were included in the study. Indications for surgery included the presence of CT-documented mass effect with or without the presence of intracranial haematoma, evidence of raised ICP on monitoring and/or deteriorating neurological status. Exclusion criteria included patients who died within 3 days after the surgery as we believe that in this period there is not enough time to develop hydrocephalus.

All patients were admitted and managed in the neurosurgical intensive care unit (NSICU) and received standard medical management of cerebral oedema. Serial CT scans of the head were acquired in all patients. Epidemiological data such as age, sex, mode and time of injury, admission GCS score, date, time and type of surgeries and post-surgical outcome and history were obtained from the Computerised Patient Record System.

CT head was reviewed to see for any ventriculomegaly and/or subdural effusion. Ventriculomegaly was defined as the presence on CT scans (in the postoperative period) of *both* of the following criteria: 1) modified Frontal Horn Index score greater than 33% (the greatest width of the frontal horns divided by the bicortical distance in the same plane), and 2) distended appearance of the anterior horns of the lateral ventricles and the enlargement of the temporal horns and third ventricle in the presence of normal or absent sulci and/or presence of periventricular lucency. Subdural effusions were classified as ipsilateral, contralateral, bilateral and interhemispheric depending on the site of craniectomy. Scans revealing ventriculomegaly were serially followed in order to assess the evolution of the hydrocephalus vis-à-vis subdural effusion and/or midline shift. Individual lesions identified on CT scans, such as diffuse brain oedema, cerebral contusions, traumatic SAH, IVH, or intra/extra-axial haemorrhagic collections, were also recorded. Data from subsequent scans were also recorded. In-hospital mortality was assessed for all patients.

## 3. Results

A total of 270 patients had undergone decompressive craniectomy in the study period. Of these 80 (26.6%) patients developed subdural effusion (Figs. 1 and 2). Males constituted 83% of these patients with the mean age of 30 years. Majority of the patients (35%) were in the 20–30 year group. The most common cause of trauma was road traffic accidents (92%) followed by falls (6%). Sixty percent patients had acute subdural haematoma followed by contusions (31.25%), diffuse brain oedema (6.25%) and bullet injury (2.5%). The admission GCS was $\leq$8 in 72.5% patients, rest being moderate head injuries (GCS 9 $\leq$ 13).

The presence of SEV was 26.62%. SEV manifested earliest in 3 days but majority of patients (52%) developed SEV in the 3rd postoperative week with a mean of 10 days. Most common site of the SEV was ipsilateral to the site of craniectomy (70%) followed by interhemispheric (47.5%) and bilateral (22.5%). The average thickness of the SEV was 14.67 mm (57.5%). It was more than 20 mm in 23.75% patients with the maximum of 39 mm in 2 patients.

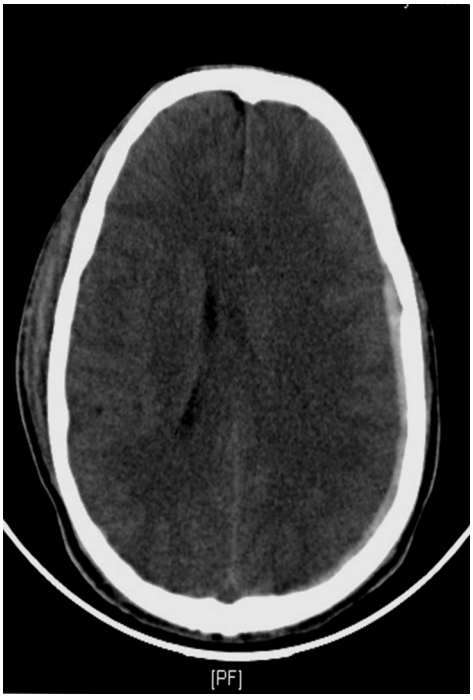


**Fig. 1 – CT scan brain showing left frontotemporoparietal acute subdural haematoma with mass effect.**

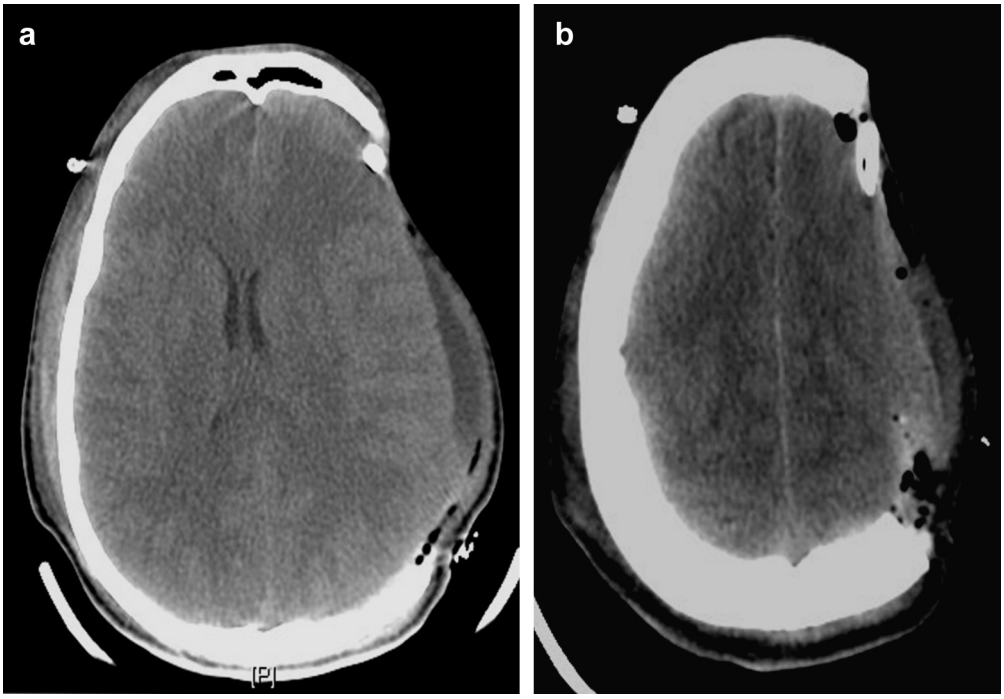


**Fig. 2 – Postoperative CT scan of the same patient 2 days after surgery showing evidence of left frontotemporoparietal decompressive craniectomy and appearance of subdural effusion (a) but no interhemispheric collection (b).**

### 3.1. *Interhemispheric hygroma (IHH) and association with ventriculomegaly*

Nearly half of the patients with SEV had interhemispheric hygroma (47.5%) ranging in thickness from 3.5 mm to 23 mm with an average width of 8.71 mm (Fig. 3). The incidence of IHH after decompressive craniectomy was 14%. In sixty percent patients the thickness was between 5 and 10 mm.

30 out of 38 patients having IHH developed ventriculomegaly (Fig. 4a and b) and the incidence in patients harbouring IHH was 79%. The mean modified Frontal Horn Index was 0.38 (range 0.318–0.412). Thirty patients out of forty two (71.39%) developed ventriculomegaly within 45 days whereas 12 patients (28.57%) developed it after 45 days (delayed ventriculomegaly). 93.3% ($n = 28$) patients developing early ventriculomegaly had associated IHH. The appearance of ventriculomegaly was followed by a compensatory decrease in the subdural effusion as well as IHH.

IHH was found to be a significant precursor of ventriculomegaly in patients with SEV and could easily predict the need of aggressive management in such patients.14 out of 42 patients with HCP (33.33%) required treatment in the form of different types of shunts [subduroperitoneal shunt (SPS), lumboperitoneal shunt (LPS), burr hole evacuation and ventriculoperitoneal shunt (VPS)]. 12 (85.71%) of such patients were also harbouring IHH with a mean of 8.7 mm (Fig. 5).

Of the various modalities of treatment, 1 patients needed burr hole evacuation, 3 had SPS, 3 had LPS and 7 had primary VP shunt. All the patients managed with SPS and burr hole evacuation needed secondary VP shunt for increasing HCP and mass effect. The patients with LPS however were lost to

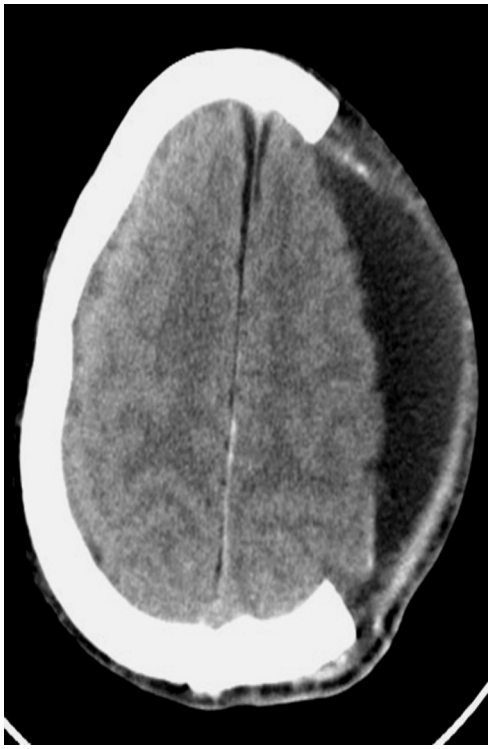

**Fig. 3 – CT scan brain taken on 9th postoperative day revealing subdural effusion and interhemispheric hygroma (IHH).**

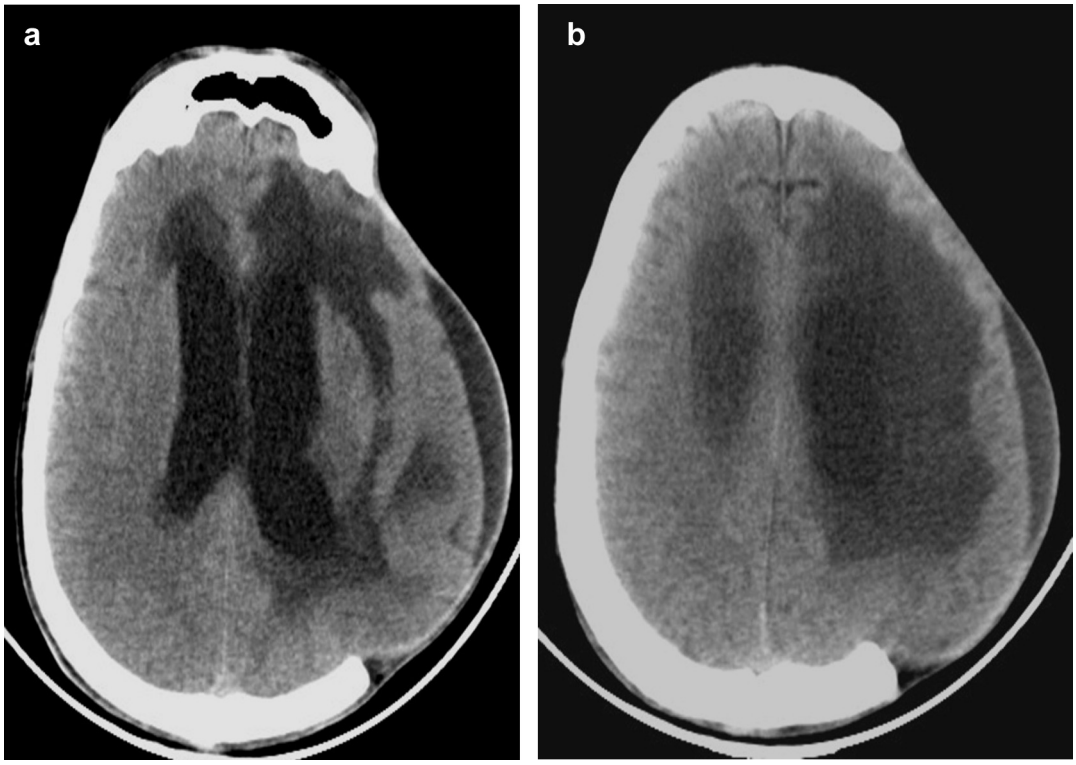


**Fig. 4 – CT scan 2 months after the surgery revealing subdural effusion with ventriculomegaly (SEV). Note the compensatory decrease in the amount of subdural effusion (a) and the IHH (b).**

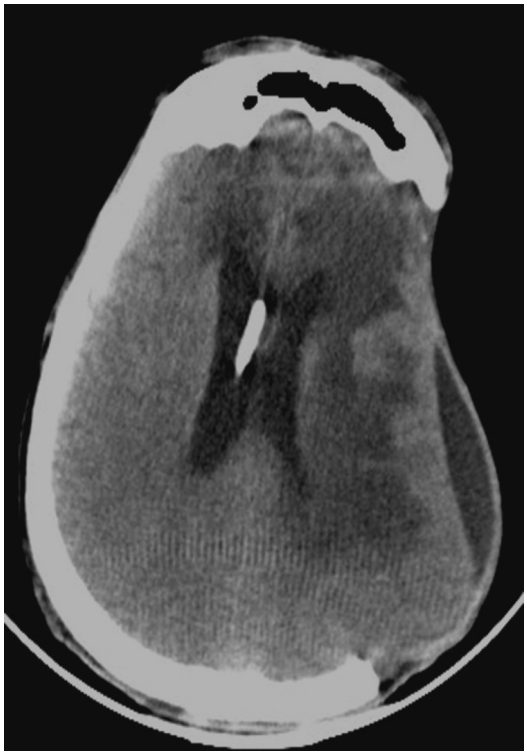

**Fig. 5 – CT scan after insertion of right parietal ventriculoperitoneal shunt. Ventriculomegaly and the effusion both have decreased after the procedure.**

follow up. All the patients who had VP shunt showed decrease in ventriculomegaly as well as IHH on radiological assessments.

### 3.2. *SEV and mortality following DC*

22 out of 80 patients died (27.8%). The last CT head done (prior to death) showed resolution of subdural effusion in 4 patients. Four patients had subdural effusion along with midline shift (range 7.32–17.9 mm) while 14 had IHH (64%) ranging from 7.5 mm to 17.8 mm (mean 10.22 mm). Thus the risk of mortality was also proportional to the width of IHH and hence it can be considered as a marker of poor outcome or mortality.

## 4. Discussion

The incidence of hydrocephalus in patients suffering traumatic head injury and requiring DC has been reported to be 0.7–86%,[8,11–13] the wide range owing to different evaluation criteria. Using radiological criteria for the diagnosis, we observed an incidence of hydrocephalus of 15.5% (42 of 270 patients). The incidence of hydrocephalus in patients developing subdural collection was very high (42 of 80 patients).

Patients undergoing decompressive craniectomy have increased chances of developing subdural collections owing to the deranged balance between CSF production, circulation and absorption. In our study, subdural effusion seen in 26.6%

of the patients. Despite it being a common entity, the ideal management of the condition remains ill defined.

Kaen et al have suggested a correlation between interhemispheric hygroma and the development of hydrocephalus.[14] In their study the IHH had preceded the development of overt hydrocephalus in more than 85% and had appeared before the development of HCP. In our series too IHH preceded the development of HCP in more than 90% patients but it appeared after the development of subdural effusion in about 80% patients.

The physiological mechanisms causing HCP after DC are yet to be determined and the probabilities can be summarized as:

1. Abnormal CSF circulation due to posttraumatic subarachnoid haemorrhage and cranial surgery particularly craniectomy.[15–19]
2. Formation of a one-way valve due to arachnoid tear, either by shear stress after TBI or surgical injury, allowing unidirectional pass of CSF.[4]
3. Leakage of serum fluid from fenestrations of small vessels on subdural neomembranes and concomitant enlargement of the subdural hygromas.[20]

Recent studies by Kaen and colleagues have proposed different phases of development of IHH and HCP such as 1) rebound phase when there is traction on the falx soon after DC leading to an expansion of the interhemispheric space and causing IHH, and a 2) hydrodynamic phase leading to the subdural collection. Analysing the serial CT scans of the patients for the evolution of SEV, and contrary to the rebound phase, we propose that the evolution of IHH is a result of decreased absorption secondary to the blockage of the arachnoid granulations with blood or its proteinaceous breakdown products seen typically after trauma or surgery. In our study the IHH was seen after the appearance of the subdural effusion in 30 of the 38 patients. Brain bulge was not a significant finding in our study (seen in only 6 out of 80 subdural effusions i.e., 7.5%) and though decompression can cause expansion of the interhemispheric space, the development of IHH cannot be attributed to it alone.

SEV occurs when the abnormal CSF circulation is combined with communication between the subdural space and the ventricles. The CSF is diverted to the subdural space because the convexity of the brain has less resistance (especially after the removal of the cranial vault in DC) compared to the ependyma of the ventricles and the formation of the subdural CSF collection requires less pressure than the ventricular enlargement.[4]

The presence of an extra-axial collection with mass effect makes the decision to treat an SEV with a V-P shunt difficult and many surgeons would argue that it is better to wait until the subdural collection has been absorbed and the hydrocephalus is established.[3] However, this practice is not without risk because shunting at a later stage might not reverse a neurological deficit. Also, dealing with the subdural collection first with a simple burr hole evacuation of the subdural effusion is not without risks; the cause of the SEV remains untreated and the patient might develop CSF leak and subsequently infection which will delay further the implantation of the VP shunt. After diagnosis of SEV has been made, we would advise to treat this condition with a VP shunt.

Poca et al have proposed the continuous monitoring of intracranial pressure (ICP) to diagnose these difficult cases of ventriculomegaly, particularly in complex cases, and it seems that high mean ICP and plateau waves are good prognostic factors for satisfactory outcome after shunt insertion.[13,17] Recently, Huh et al [21] suggested measuring the subdural pressure using a manometer intraoperatively, before opening the dura mater, in patients with subdural collections and ventriculomegaly. Both methods will be helpful in managing patients with SEV because there is no need for a lumbar puncture which carries the risk of increasing the size of the subdural collection in cases of a misdiagnosed subdural hygroma.

In patients with SEV after decompressive craniectomy, the placement of VP Shunt may be sufficient to take care of both the hydrocephalus and subdural collection. Other authors have also suggested early cranioplasty for correction of CSF hydrodynamics after decompressive craniectomy particularly in cases of the "syndrome of the trephine".[17–19,22–24]

## 5. Conclusion

Our findings confirm that posttraumatic hydrocephalus is a common complication following DC. The physiological mechanism by which hydrocephalus develops after DC remains to be determined. Contrary to common perception, SEV has a very high incidence (26.6% in our study) in patients who required DC following TBI. Our study shows that IHH is an important prognostic marker for the need of CSF diversion in these patients.

## Conflicts of interest

All authors have none to declare.

## REFERENCES

1. Yang XJ, Hong GL, Su SB, Yang SY. Complications induced by decompressive craniectomies after traumatic brain injury. *Chin J Traumatol.* 2003;6:99–103.
2. Tzerakis N, Orphanides G, Antoniou E, Sioutos PJ, Lafazanos S, Seretis A. Subdural effusions with hydrocephalus after severe head injury: successful treatment with ventriculoperitoneal shunt placement: report of three adult cases. *Case Rep Med.* 2010, 743784.
3. Yoshimoto Y, Wakai S, Hamano M. External hydrocephalus after aneurysm surgery: paradoxical response to ventricular shunting. *J Neurosurg.* 1998;88:485–489.
4. Kawaguchi T, Fujita S, Hosoda S, Shibata Y, Komatsu H, Tamaki N. Treatment of subdural effusion with hydrocephalus after ruptured intracranial aneurysm clipping. *Neurosurg.* 1998;43:1033–1039.
5. Ment LR, Duncan CC, Geehr R. Benign enlargement of the subarachnoïd spaces in the infant. *J Neurosurg.* 1981;54:504.
6. Robertson WG, Gomez MR. External hydrocephalus: early finding in congenital communicating hydrocephalus. *Arch Neurol.* 1978;35:541.

7. Cardoso ER, Schubert R. External hydrocephalus in adults. Report of three cases. *J Neurosurg*. 1996;85:1143–1147.
8. Choi I, Park HK, Chang JC, Cho SJ, Choi SK, Byun BJ. Clinical factors for the development of posttraumatic hydrocephalus after decompressive craniectomy. *J Korean Neurosurg Soc*. 2008;43:227–231.
9. Escosa-Bage, Sola RG. Physiopathology of adult onset external hydrocephalus. *Rev Neurol*. 2002;35:141–144.
10. Kilincer C, Simsek M, Hamamcioglu K, Hicdonmez T, Cobanoglu S. Contralateral subdural effusion after aneurysm surgery and decompressive craniectomy: case report and review of the literature. *Clin Neurol Neurosurg*. 2005;107:412–416.
11. Licata C, Cristofori L, Gambin R, Vivenza C, Turazzi S. Posttraumatic hydrocephalus. *J Neurosurg Sci*. 2001;45: 141–149.
12. Marmarou A, Foda MA, Bandoh K, et al. Posttraumatic ventriculomegaly: hydrocephalus or atrophy? A new approach for diagnosis using CSF dynamics. *J Neurosurg*. 1996;85:1026–1035.
13. Poca MA, Sahuquillo J, Mataró M, Benejam B, Arikan F, Báguena M. Ventricular enlargement after moderate or severe head injury: a frequent and neglected problem. *J Neurotrauma*. 2005;22:1303–1310.
14. Kaen A, Jimenez-Roldan Luis, Alday R, Gomez Pedro A, Lagares A, Alén José Fernández. Interhemispheric hygroma after decompressive craniectomy: does it predict posttraumatic hydrocephalus? *J Neurosurg*. 2010;113: 1287–1293.
15. Aarabi B, Chesler D, Maulucci C. Dynamics of subdural hygroma following decompressive craniectomy: a comparative study. *Neurosurg Focus*. 2009;26:E8.
16. Kilincer C, Hamamcioglu MK. Surgical complications of decompressive craniectomy for head trauma. *Acta Neurochir*. 2010;152:557–558.
17. Waziri A, Fusco D, Mayer SA, et al. Postoperative hydrocephalus in patients undergoing decompressive hemicraniectomy for ischemic or hemorrhagic stroke. *Neurosurgery*. 2007;61:489–493.
18. Yang XF, Wen XF, Shen F. Surgical complications secondary to decompressive craniectomy in patients with head injury: a series of 108 consecutive cases. *Acta Neurochir*. 2008;150: 1241–1248.
19. Yang XF, Wen L, Gong JB, Zhan RY. Subdural effusion secondary to decompressive craniectomy in patients with severe traumatic brain injury. *Acta Neurochir*. 2010;152: 555–556.
20. Hasegawa M, Yamashima T, Yamashita J. Traumatic subdural hygroma: pathology and meningeal enhancement on magnetic resonance imaging. *Neurosurg*. 1992;31:580–585.
21. Huh PW, Yoo S, Cho KS. Diagnostic method for differentiating external hydrocephalus from simple subdural hygroma. *J Neurosurg*. 2006;105:65–70.
22. Carvi MN, Nievas Y, Höllerhage HG. Early combined cranioplasty and programmable shunt in patients with skull bone defects and CSF-circulation disorders. *Neurol Res*. 2006;28:139–144.
23. Fodstad H, Love JA, Ekstedt J. Effect of cranioplasty on cerebrospinal fluid hydrodynamics in patients with syndrome of the trephined. *Acta Neurochir*. 1984;70:21–30.
24. Liang W, Xiaofeng Y, Weiguo L. Cranioplasty of large cranial defect at an early stage after decompressive craniectomy performed for severe head trauma. *J Craniofac Surg*. 2007;18:526–532.