Symptomatic contralateral subdural hygromas after decompressive craniectomy: plausible causes and management protocols

Pravin Salunke, MCh, Ravi Garg, MCh, Ankur Kapoor, MS, Rajesh Chhabra, MCh, and Kanchan K. Mukherjee, MCh

Department of Neurosurgery, Postgraduate Institute of Medical Education and Research, Chandigarh, India

OBJECT Contralateral subdural hygromas are occasionally observed after decompressive craniectomies (DCs). Some of these hygromas are symptomatic, and the etiology and management of these symptomatic contralateral subdural collections (CLSDCs) present surgical challenges. The authors share their experience with managing symptomatic CLSDCs after a DC.

METHODS During a 10-month period, 306 patients underwent a DC. Of these patients, 266 had a head injury, 25 a middle cerebral artery infarction (that is, a thrombotic stroke), and 15 an infarction due to a vasospasm (resulting from an aneurysmal subarachnoid hemorrhage [SAH]). Seventeen patients (15 with a head injury and 2 with an SAH) developed a CLSDC, and 7 of these patients showed overt symptoms of the fluid collection. These patients were treated with a trial intervention consisting of bur hole drainage followed by cranioplasty. If required, a ventriculo- or thecoperitoneal shunt was inserted at a later time.

RESULTS Seven patients developed a symptomatic CLSDC after a DC, 6 of whom had a head injury and 1 had an SAH. The average length of time between the DC and CLSDC formation was 24 days. Fluid drainage via a bur hole was attempted in the first 5 patients. However, symptoms in these patients improved only temporarily. All 7 patients (including the 5 in whom the bur hole drainage had failed and 2 directly after the DC) underwent a cranioplasty, and the CLSDC resolved in all of these patients. The average time it took for the CLSDC to resolve after the cranioplasty was 34 days. Three patients developed hydrocephalus after the cranioplasty, requiring a diversion procedure, and 1 patient contracted meningitis and died.

CONCLUSIONS Arachnoid tears and blockage of arachnoid villi appear to be the underlying causes of a CLSDC. The absence of sufficient fluid pressure required for CSF absorption after a DC further aggravates such fluid collections. Underlying hydrocephalus may appear as subdural collections in some patients after the DC. Bur hole drainage appears to be only a temporary measure and leads to recurrence of a CLSDC. Therefore, cranioplasty is the definitive treatment for such collections and, if performed early, may even avert CLSDC formation. A temporary ventriculostomy or an external lumbar drainage may be added to aid the cranioplasty and may be removed postoperatively. Ventriculoperitoneal or thecoperitoneal shunting may be required for patients in whom a hydrocephalus manifests after cranioplasty and underlies the CLSDC.

http://thejns.org/doi/abs/10.3171/2014.10.JNS14780

KEY WORDS decompressive craniectomy; contralateral subdural collections; cranioplasty; trauma

SUBDURAL hygromas result from disorders in CSF flow and are a complication of decompressive craniectomies (DCs), with an incidence of approximately 21%–50%. Most of these hygromas are ipsilateral, and the majority of them (70%–90%) resolve spontaneously. One type of hygroma, contralateral subdural collection (CLSDC) arising from DCs, is encountered less often but is more likely to be symptomatic. The management of symptomatic CLSDCs represents a clinical challenge, because they affect the outcome of the DCs and therefore need to be treated aggressively. The literature on managing CLSDCs is limited to case reports and short series. The authors of these reports have described both symptomatic and asymptomatic CSF flow.
collections, have observed that symptomatic collections are rare, and have noted that most cases respond well to conservative management. Bur hole drainage and subdural-peritoneal shunting with or without cranioplasty have been suggested for those patients whose lesions do not respond to these conservative approaches. Because of the paucity of data, it remains unclear how best to manage such collections. We share our experience in managing symptomatic CLSDCs after DC. Because treatment of these collections with bur hole drainage failed to resolve the CLSDCs in our cases, we attempted to establish a different treatment strategy for these recalcitrant fluid collections. Although we present no primary evidence, we propose plausible causes for the formation of CLSDCs on the basis of indirect evidence.

Methods

Patient Cohort

In a 10-month period between August 2012 and May 2013, 306 patients underwent wide DCs at our center for intractable, raised intracranial pressure (ICP) after a head injury, infarctions with edema due to subarachnoid hemorrhages (SAHs), or malignant arterial infarction (that is, thrombotic stroke) with a mass effect. Of these patients, 88 developed subdural CSF collections in the follow-up period after DC.

Clinical and Radiological Profiles

Of these 88 patients, 17 had a CLSDC, 10 of whom had minor symptoms (asymptomatic or a small wound bulge). These collections were detected on routine follow-up CT scans taken at weekly intervals, initially for 1 month and then later at 3–4 week intervals. The remaining 7 patients had shown initial clinical improvement after a DC, but exhibited worsening symptoms a few days later. This worsening manifested in patients who were conscious after DC as progressive hemiparesis on the side of the DC (which was previously normal), as decreased sensorium (worsening of Glasgow Coma Scale scores after the DC had initially improved scores), as headache and vomiting or incontinence, or as a significant wound bulge. A radiographic examination indicated the appearance of a new CLSDC with a significant mass effect (that is, showing a midline shift of >8 mm). These collections were not detected on a CT scan taken soon after the DC. Patients with intracranial pathological changes requiring DC often have significant long-term neurological deficits for multiple reasons.

Management of Contralateral Collections After DC

This study included only the 7 patients who had a symptomatic CLSDC. The first 5 patients were studied retrospectively and the remaining 2 prospectively. Informed consent was obtained from the patients before any procedure. Figure 1 shows the treatment algorithm we followed. Patients with a symptomatic CLSDC underwent bur hole drainage, and those whose condition did not improve in response to this procedure underwent cranioplasty. During the cranioplasty, the subdural collection was retapped, if required, to achieve a relaxation of the brain tissue, which aided the procedure. After the cranioplasty, if the patient developed hydrocephalus, a CSF diversion procedure was added. The clinical and radiographic outcomes were evaluated at regular 3- to 4-week intervals.

Results

Patient Profile and Incidence of CLSDCs

Of 306 patients who underwent a DC, 266 had a head injury, 15 had infarctions with a mass effect due to a SAH, and 25 had an infarction of the middle cerebral artery. In total, 17 patients developed a CLSDC, of whom 3 had undergone a DC because of SAH-related infarctions and 14 because of head trauma. None of the patients who underwent a DC to treat arterial infarctions (that is, strokes) developed these collections.

Contralateral collections became symptomatic in 7 (6 men and 1 woman) of these 17 patients, and their incidence was 2.2% in patients who had a head injury and 6.6% in patients developing infarctions after SAHs. Of these 7 patients, 6 had been treated for a head injury and 1 for an infarction after an SAH.

The demographic profiles, diagnoses, time periods, and outcomes in the 7 patients are shown in Table 1. The average age of these patients was 40 years (range 27–52 years).

Clinical Features of CLSDCs

The average time from the DC to the development of a CLSDC was 23.8 days (range 5–62 days). These CLSDCs coincided with symptoms of hemiparesis (contralateral to the side of the CLSDC) in 5 patients, significant wound bulging in 4 patients, headache in 1 patient, and decreased sensorium in 2 patients.

Management and Outcomes of the Symptomatic CLSDCs

The first 5 patients underwent bur hole drainage for the symptomatic CLSDCs, which led only to transient improvement. A bur hole tap was repeated in 2 patients without much improvement. The last 2 patients underwent cranioplasty directly, without a bur hole drainage. All 7 patients showed resolution of the CLSDC after the cranioplasty (Fig. 2). However, only 4 of these patients showed progressive improvement. The remaining 3 patients exhibited initial clinical improvement, but despite radiographically confirmed resolution of CLSDCs, these 3 patients had symptoms of hydrocephalus, 2 of whom underwent ventriculoperitoneal shunting and 1 of whom underwent thecoperitoneal shunting. These patients showed both clinical and radiographically confirmed improvement after surgery for CSF diversion (Fig. 3). The average time from the cranioplasty to resolution of the CLSDC was 34 days (range 1–90 days).

All patients improved after the cranioplasty irrespective of the CSF diversions. One patient died after contracting meningitis in the follow-up period. This patient had shown initial clinical improvement and resolution of the CLSDC after the cranioplasty. However, 6 weeks after the cranioplasty, the patient was readmitted for fever and
an altered sensorium. A CT scan of the head showed no fluid collections or hydrocephalus, and examination of the patient's CSF obtained with lumbar puncture indicated a meningitis infection. Despite treatment with antibiotics, the patient died of the infection.

**Discussion**

Decompressive craniectomy is now considered an important procedure for the management of refractory intracranial hypertension. It is usually performed in patients who have a head injury or ischemic infarctions. However, a DC is not without complications,

and disorders resulting from abnormal CSF circulation, such as subdural hygromas or hydrocephalus, are common after a DC.

**Subdural Fluid Collections After DC**

Subdural CSF collections are a well-known complication of DC and have an incidence of 21%–50%. Most of these collections are ipsilateral to the DC, are often asymptomatic, and resolve spontaneously. Therefore, surgical intervention is required only in 8%–30% of patients who have subdural CSF collections. Subdural CSF collections appear in the 1st week after the DC, reach their peak volume by Week 4, and disappear by approximately Week 17. Their pathogenesis is unclear. Multiple theories have been proposed to explain their occurrence and maintenance. These hypotheses include the following: 1) a tear at the subarachnoid-dural interface, produced either by shear stress generated by kinetic energy or by an injury during surgery, creates a unidirectional flow of the CSF (due to a valve effect) that generates and maintains the collection; 2) a vascular or parenchymal injury produces an effusion to the subdural space; or 3) an increased arachnoid permeability due to physical disruption.

![Algorithm for managing CLSDCs after DCs for SAHs or trauma](image-url)
TABLE 1. Details of the patients showing symptoms of a CLSDC after DC

<table>
<thead>
<tr>
<th>Pt No.</th>
<th>Age (yrs)</th>
<th>Initial Diagnosis Leading to DC</th>
<th>Interval Btwn Injury/Insult &amp; DC (days)</th>
<th>Initial GCS Scores</th>
<th>Side of DC</th>
<th>Discharge or Postop GCS Scores &amp; Presentation</th>
<th>CLSDC Symptoms &amp; Presentation</th>
<th>Time From DC to CLSDC (days)</th>
<th>Time Btwn Bur Hole Drainage &amp; Cranioplasty (days)</th>
<th>CSF Diversion</th>
<th>Time of Resolution of CLSD After Cranioplasty (days)</th>
<th>Outcome of Cranioplasty</th>
<th>Length of FU (mos)</th>
<th>GOS Score at Last FU</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>52</td>
<td>Rt MCA aneurysm w/ rt MCA infarct</td>
<td>2</td>
<td>E₂VTM₅ w/ lt hemiplegia</td>
<td>Rt</td>
<td>E₂VTM₅ w/ lt hemiplegia &amp; rt hemiparesis</td>
<td>E₂VTM₅ w/ lt hemiplegia &amp; rt hemiparesis</td>
<td>5</td>
<td>2</td>
<td>No</td>
<td>60 E₂VTM₅ w/ lt hemiplegia &amp; rt hemiparesis</td>
<td>E₂VTM₅ w/ lt hemiparesis improved</td>
<td>14</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>35</td>
<td>Lt temporal contusion w/ lt acute SDH</td>
<td>1</td>
<td>E₁VAM₅ w/ rt hemiparesis</td>
<td>Lt</td>
<td>E₁VAM₅ w/ rt hemiparesis</td>
<td>Same scores as at discharge w/ wound bulge &amp; lt hemiparesis</td>
<td>15</td>
<td>30</td>
<td>Yes</td>
<td>1 E₁VAM₅ w/ lt side strength improved</td>
<td></td>
<td>15</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>43</td>
<td>Rt side acute SDH</td>
<td>4</td>
<td>E₁VTM₅ w/ lt hemiparesis</td>
<td>Rt</td>
<td>E₁VTM₅ w/ lt hemiparesis</td>
<td>Same scores as at discharge w/ rt hemiparesis</td>
<td>18</td>
<td>16</td>
<td>Yes</td>
<td>10 E₁VAM₅ w/ rt side strength improved</td>
<td></td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>45</td>
<td>Lt side acute SDH</td>
<td>5</td>
<td>E₁VTM₅ w/ rt hemiparesis</td>
<td>Lt</td>
<td>E₁VTM₅ w/ rt hemiparesis</td>
<td>Same scores as at discharge w/ lt hemiparesis</td>
<td>17</td>
<td>56</td>
<td>No</td>
<td>45 Initial improvement but died later from meningitis</td>
<td></td>
<td>3</td>
<td>NA</td>
</tr>
<tr>
<td>5</td>
<td>30</td>
<td>Lt side acute SDH w/ frontal contusions</td>
<td>1</td>
<td>E₁VTM₅ w/ rt hemiparesis</td>
<td>Lt</td>
<td>E₁VTM₅ w/ rt hemiparesis</td>
<td>E₁VTM₅ w/ lt hemiparesis w/ wound bulge</td>
<td>62</td>
<td>2</td>
<td>Yes</td>
<td>30 E₁VTM₅ w/ lt hemiparesis improved</td>
<td></td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>27</td>
<td>Lt side acute SDH</td>
<td>1</td>
<td>E₁VAM₅ w/ rt hemiparesis</td>
<td>Lt</td>
<td>E₁VAM₅ w/ rt hemiparesis</td>
<td>Headache w/ wound bulge &amp; partial improvement</td>
<td>30</td>
<td>NA</td>
<td>No</td>
<td>90 E₁VAM₅ w/ subtle hemiparesis</td>
<td></td>
<td>18</td>
<td>5</td>
</tr>
<tr>
<td>7</td>
<td>48</td>
<td>Lt side acute SDH w/ frontal contusions</td>
<td>1</td>
<td>E₁VAM₅ w/ rt hemiparesis</td>
<td>Lt</td>
<td>E₁VAM₅ w/ rt hemiparesis</td>
<td>Same scores as at discharge w/ wound bulge</td>
<td>45</td>
<td>NA</td>
<td>No</td>
<td>1 E₁VAM₅ w/ min rt hemiparesis</td>
<td></td>
<td>12</td>
<td>3</td>
</tr>
</tbody>
</table>

A = aphasic; E = eye; FU = follow-up; GCS = Glasgow Coma Scale; GOS = Glasgow Outcome Scale; M = movement; MCA = middle cerebral artery; min = minimal; NA = not applicable; Pt = patient; SDH = subdural hematoma; T = tracheostomized; V = voice.

* Patient 7 was a woman and all other patients were men; Patients 1–5 were treated with bur hole drainage, which was not used for Patients 6 and 7. VA or VT denote that patients who were aphasic or tracheostomized, respectively, which prohibited an assessment of their voice-related GCS scores.
† Except for Patient 1, these were the GCS scores at the time of discharge after the DC. Patient 1 developed a CLSDC in the period during which he was treated with DC; therefore, the scores for this patient indicate his postoperative GCS scores.
or higher transmembrane pressure produces and sustains the collection.

Contralateral subdural fluid collections are more often symptomatic than ipsilateral fluid collections and pose a different type of challenge. Several mechanisms have been proposed to be involved in the development of CLSDCs, including the following: 1) a rapid decrease in the ICP and an outward herniation may create a pressure gradient between the 2 dural hemispheres; this gradient may cause the contralateral subdural space to enlarge and the accumulation of effusion, especially in cases of an initial rupture of the arachnoid layer after a head trauma.6,10,14 2) A disturbed CSF circulation, especially diminished CSF absorption, may increase the risk for accumulation of the effusion through the torn portion of the arachnoid layer.16 3) Shrinkage of the ipsilateral region of the brain due to intraoperative tissue retraction and failure of the brain to readapt to the intracranial space may cause a pressure gradient between the 2 hemispheres and may also lead to a brain shift. This shift may play an important role in the enlargement of the contralateral subdural space as well as in the accumulation of subdural effusions.5,10

Such fluid collections were never observed when we used DC to treat an ischemic stroke. Kilincer and Hamamcioglu have highlighted that unlike for traumatic brain injuries, subdural collections are extremely rare after a DC for large hemispherical infarctions.4 This clearly indicates that the involvement of the arachnoid layer (that is, its damage due to trauma or via blockage of the arachnoid villi due to an SAH) is essential for the formation of CLSDCs.

Options and Rationales for CLSDC Treatment

Treatment options for symptomatic fluid collections include bur hole drainage, subduroperitoneal shunting with or without cranioplasty, and CSF diversions.5,11,14 However, studies of the use of these approaches are limited to case reports and short case series, and the number of patients in whom a symptomatic CLSDC was managed is very small.5,11,14

Bur hole drainage has been suggested for treating CSF collections including CLSDCs.5,11,12,14 However, bur hole drainage gave mixed results in our series, because it appeared to provide only temporary relief and did not prevent recurrence of the CLSDC. The reason for this failure of providing a lasting favorable outcome may lie in the fact that a CSF pressure of 2–5 cm H$_2$O is required to open the arachnoid villi because an open villus is essential for sufficient CSF absorption.13 A craniectomy defect may lead to a loss of the normal dicrotic ICP waveform required to keep the arachnoid villi open, and a flattening of the waveform in such cases indicates failure of CSF reabsorption.13 This lack of CSF reabsorption is probably the reason that bur hole drainage in our patients resulted only in a transient improvement both clinically and radiographically. Restoration of a normal dicrotic ICP waveform appears to be necessary to resolve CSF collections such as CLSDCs. According to the aforementioned model of impaired CSF reabsorption, a bur hole alone is probably insufficient to restore the CSF pressure to levels sufficient for its absorption. Accordingly, bur hole drainage does not appear to be a valid option for treating CLSDCs.

Our data suggest cranioplasty to be a cornerstone for treating fluid collections such as CLSDCs. Most of the CLSDCs resolved after the cranioplasty, but a few required additional CSF diversion. Cranioplasty restores the dicrotic ICP waveform, helping to facilitate sufficient

![Image](69x469 to 525x721)

**Fig. 2.** Serial noncontrast CT scans of the head of a patient who underwent a DC for a head injury. Note the appearance of a CLSDC on Day 45, which was symptomatic. The Day 46 scan indicates resolution of the CLSDC after cranioplasty, and symptoms also improved.
CSF absorption. This indicates that when the cranioplasty is performed early, it probably prevents the formation of CLSDCs. The timing of a cranioplasty after a DC has been critically discussed. The older practice of delaying cranioplasty to 3–6 months after a DC seems neither to lower postcranioplasty infection rates nor to avert the need for CSF diversion. In fact, Beauchamp et al. have suggested that cranioplasty be performed as soon as CT scans show resolution of brain swelling outside of the cranial vault. This resolution occurs as early as 2 weeks postcraniectomy. Thus, cranioplasty as soon as the swelling diminishes after a DC likely avoids complications such as CLSDCs.

Because of persistent brain swelling, early cranioplasty may not always be possible, especially in the absence of clinical improvement. In such cases, a CLSDC may develop. Our data suggest that urgent cranioplasty may help in these cases, possibly by restoring the dicrotic ICP waveform. However, cranioplasty may be difficult if a wound bulge is present after the CLSDC. Because the underlying cause of a CLSDC may be a hydrocephalus (see below), placing a temporary ventriculostomy during the surgery to avoid infection may also relax the brain, which aids the cranioplasty. The ventriculostomy placement may be difficult because of small ventricles, and a good alternative is a placement of an external lumbar drain. This placement is relatively safe even in the presence of brain swelling after a DC because a paradoxical herniation after lumbar drainage in the setting of a DC is rare. The ventricular or lumbar drain may be removed after the cranioplasty, and a permanent CSF diversion (such as a shunt) may be added if a hydrocephalus appears later.

The need for CSF diversion in 3 of our patients suggested underlying hydrocephalus as another cause of the CLSDCs. It is possible that a trauma or subarachnoid bleeding caused an abnormal communication between ventricles and the subarachnoid space in either hemisphere. The flattening of the dicrotic waveform of the CSF pressure creates a low resistance, pushing the CSF from the ventricles to the subarachnoid space through these abnormal communications. Cranioplasty restores the CSF pressure waves and thus increases resistance to such ab-

**FIG. 3.** Serial noncontrast CT scans of the head of a patient who also underwent a DC for a head injury. Note the appearance of CLSDC (on Day 15) and its resolution after cranioplasty (on Day 45). Five months later, the patient developed hydrocephalus for which a ventriculoperitoneal shunt was required.
normal pathways. Hydrocephalus may become evident on radiographs and may be the reason for the subdural collections.\(^5\)

Some authors have suggested placement of a subduraloperitoneal shunt for subdural collections.\(^5,14\) However, these collections are under low pressure (that is, of only 0–5 mm Hg), and placement of these shunts alone may not be helpful. According to Paredes et al. and Waziri et al., increasing the ICP would increase the hydrostatic pressure that forces fluid into the vessels.\(^6,13\) As a result, there is a lack of a pressure gradient keeping the subduraloperitoneal shunt patent, which may increase the risk for an infection.

On the basis of our experience, we suggest a management algorithm for patients with subdural CSF collections after a DC (Fig. 4). The small number of patients in the present study weakens the utility of this algorithm. Nevertheless, this algorithm provides suggestions for managing CLSDCs, which are fortunately not very common but do require aggressive management.

**Conclusions**

An arachnoid tear and blockage of arachnoid villi appear to produce a CLSDC. The absence of pressure required for CSF absorption after a DC further aggravates such CSF collections. Underlying hydrocephalus may cause subdural collections in a few patients after a DC. The etiology of the CLSDCs proposed here is merely a hypothesis for which we do not have any direct evidence.

Cranioplasty as soon as the swelling diminishes after a DC may avert the CLSDCs. However, if a patient shows symptoms of a CLSDC earlier, urgent cranioplasty seems to be the definitive treatment choice. Bur hole drainage appears to be only a temporary measure, leading to recurrence of a CLSDC. A temporary CSF diversion (for example, a ventriculostomy or external lumbar drainage) may be added if the brain is tense during cranioplasty. A ventriculo- or thecoperitoneal shunt may be required postcranioplasty in cases in which hydrocephalus is the underlying cause of a CLSDC.

**References**


Author Contributions
Conception and design: Salunke. Acquisition of data: Salunke, Garg, Kapoor. Analysis and interpretation of data: Salunke. Drafting the article: Salunke, Garg, Kapoor. Critically revising the article: Chhabra, Mukherjee.

Correspondence
Pravin Salunke, Department of Neurosurgery, PGIMER, Sector 12, Chandigarh 160012, India. email: drpravin_salunke@yahoo.co.uk.