Clinical Study
Resolution of extra-axial collections after decompressive craniectomy for ischemic stroke

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A B S T R A C T
Extra-axial fluid collections are known consequences of decompressive hemicraniectomy. Studies have examined these collections and their management. We retrospectively reviewed 12 consecutive patients who underwent decompressive hemicraniectomy for the treatment of malignant cerebral edema after infarction and evaluated the evolution, resolution and treatment of post-operative extra-axial fluid collections. All patients underwent standard-sized frontotemporoparietal hemicraniectomy with duraplasty as treatment for medically intractable malignant cerebral edema at an average of 3 days after the stroke (median 2 days). Their 30-day mortality was 25%. Three patients developed some extra-axial fluid collections after craniectomy: two patients developed the collections early in their post-operative course, 3 days and 5 days after the craniectomy. Both experienced spontaneous resolution of the collections without corrective cranioplasty or shunt placement at 34 days and 58 days after surgery. The third patient developed a collection 55 days after the operation related to a subgaleal bacterial infection. In the final analysis, 18% of patients developed extra-axial collections and all resolved spontaneously. The incidence of extra-axial collections after decompressive hemicraniectomy following ischemic stroke was lower in our retrospective series than has been reported by others. The collections resolved spontaneously, suggesting that early anticipatory, corrective treatment with cerebrospinal fluid diversion or cranioplasty may not be warranted.

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1. Introduction
Decompressive craniectomy is a life-saving procedure with benefits that have been increasingly recognized in patients with severe head injury,1 aneurysmal subarachnoid hemorrhage2 and malignant edema from middle cerebral artery (MCA) territory infarction.3,4 The last of these indications has been supported by three major European prospective trials: DECIMAL,5 DESTINY6 and HAMLET.7 While the benefits from decompressive hemicraniectomy have been well described, the complications resulting from this increasingly utilized procedure are less well studied. Some of these complications represent dynamic and unique post-operative clinical entities for which optimal management is uncertain. An enhanced understanding of these events is important in light of the increasing use of this procedure.

Sinking flap syndrome after decompressive hemicraniectomy for malignant MCA stroke is one such delayed complication. It generally occurs 3–5 months after surgery and may lead to headache, midbrain syndrome and paradoxical herniation.7 The development of extra-axial fluid collections, typically arising in the acute post-operational period, is another possible complication of this surgery. These extra-axial collections have been referred to as subdural hygromas,8 subdural fluid collections9 or external hydrocephalus.10 All of these descriptions have an implied etiological connection to hydrocephalus. Furthermore, some investigators have suggested that post-craniectomy collections should be treated with permanent cerebrospinal fluid (CSF) diversion by shunting, in the same manner that spontaneous subdural hygromas or hydrocephalus are treated.11 The clinical significance and management paradigms of these post-hemicraniectomy extra-axial collections remain unclear, as does the necessity of operative intervention to treat them.

2. Methods
We retrospectively identified consecutive patients who underwent decompressive hemicraniectomy for malignant cerebral edema secondary to MCA infarction in a 30-month period between 1 July 2007 and 31 December 2009. The Brigham and Women’s Hospital Institutional Review Board approved the study. Twelve patients who met these criteria were identified and their demographic features, surgical procedures, imaging studies, 30-day mortality and post-operative complications were extracted.
We identified post-operative extra-axial collections and reviewed the timing of any CSF shunting procedure or cranioplasty surgeries. No patients had hydrocephalus at the time of craniectomy. In our experience, extra-axial collections do not develop prior to the third day after the operation in hemicraniectomy patients, making inclusion of patients who did not reach this milestone inappropriate. Therefore, all patients who died prior to the third day were excluded from our analysis.

All patients were admitted to the Neurological Intensive Care Unit by our stroke service after diagnosis of MCA territory infarction and suspicion for increased intracranial pressure (ICP). Medical therapy for increased ICP was initiated by a protocol of head of bed elevation, sedation, hypertonic agents (20% mannitol and/or 23.4% saline) and hyperventilation to optimize the partial pressure of carbon dioxide (pCO₂; goal 30–35 mmHg). Close clinical observation was carried out, and any deterioration in the level of alertness prompted the consideration for a decompressive hemicraniectomy. The decision to proceed with surgery was made jointly by the Neuro Critical Care team and the neurosurgeon, based on either deteriorating clinical exam or a worsening mass effect from the edematous ischemic brain as visualized on CT scan, or both developments occurring simultaneously. All patients received standard frontotemporalpared cranioectomies for bony decompression and wide durotomy. Temporal lobectomies were not routinely performed. Gelfilm (Pfizer, New York, NY, USA) was placed in the epidural space to allow for easier future dissection of tissue planes during cranioplasty and Duraplasty (Codman and Shurtleff, Raynham, MA, USA) was used for duraplasty. Dural closures were not water-tight. Intraparenchymal ICP monitors or ventriculostomy drains were placed at the discretion of the surgeon, based on the degree of swelling of the brain at closure. All patients received immediate post-operative CT scans, and these were reviewed to determine the presence or absence of immediate post-operative extra-axial collections.

Cranioectomies were performed with either the patient’s autologous bone flap or with a synthetic bone flap (Synthes, West Chester, PA, USA). The purpose of cranioplasty was for cosmetic as well as protective reasons. In the period prior to cranioplasty, all patients were fitted with helmets. Patients who required shunting for underlying hydrocephalus received ventriculoperitoneal (VPS) or lumboperitoneal (LPS) shunts for CSF diversion.

The presence and resolution of extra-axial collections was determined by the authors after review of CT scans. There is no established guideline to define the minimum thickness or volume of these extra-axial collections. We defined them as fluid collections greater than 5 mm in thickness with densities consistent with CSF, rather than blood from post-operative hematomas. The timing of resolution of the collections was defined as the time to the first CT scan showing disappearance of the fluid.

### 3. Results

Over 30 months, 12 patients were identified for review (Table 1). There were eight women and four men with an average age of 57 years (range 42–72 years). The mean time from stroke to decompressive craniectomy was 3 days (range 1–8 days). The etiology causing the medically intractable cerebral edema in all 12 patients was an MCA territory infarction, with three patients having an associated anterior cerebral artery (ACA) territory infarction (25%). The strokes occurred in the right hemisphere in nine patients (75%) and in the left hemisphere in the remaining three patients (25%). ICP monitoring was performed in two of the 12 patients (16.7%) with a fiberoptic intraparenchymal monitor in one and ventriculostomy catheter in the other.

Three patients died within 30 days of surgery. The first, patient 9, developed an immediate post-operative epidural hematoma requiring re-operation with temporal lobectomy. The family withdrew care 2 days after the initial surgery. This patient was excluded from our final analysis as per our protocol, given that the patient died prior to day 3 after craniectomy. Therefore, 11 total patients were included in the final analysis. The second, patient 4, had intractably elevated ICP and uncal herniation despite craniectomy, after which the family asked that care be withdrawn 4 days post-operatively. The third, patient 5, had minimal neurological recovery after decompressive surgery and in the setting of a recent complicated cardiac valve repair, urinary tract infection, pneumonia and thrombocytopenia, care was withdrawn 30 days after her craniectomy.

Extra-axial CSF collections, as previously defined, ipsilateral to the craniectomy defect were identified in two of 11 patients; both appearing within 5 days of surgery. In patients 6 and 10 the collections appeared on post-operative days 3 and 5, respectively (Figs. 1 and 2). These patients remained neurologically stable and no treatments for the collections were initiated. The collections resolved entirely as judged from the first available CT scans on or before 58 days after surgery in Patient 6 and 34 days after surgery in

### Table 1

Demographics and outcomes of patients who underwent decompressive craniectomy for ischemic stroke showing resolution of extra-axial collections

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Side of lesion</th>
<th>Lesion territory</th>
<th>Diagnosis to craniectomy (days)</th>
<th>Time to death if &lt;30 days (days)</th>
<th>Time to extra-axial collection (days)</th>
<th>Time to resolution of collection (days)</th>
<th>Time to cranioplasty (days)</th>
<th>Time to shunt (days) (type)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>46</td>
<td>F</td>
<td>Right</td>
<td>MCA</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>386 (VPS)</td>
</tr>
<tr>
<td>2</td>
<td>53</td>
<td>F</td>
<td>Right</td>
<td>MCA</td>
<td>8</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>138</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>68</td>
<td>F</td>
<td>Right</td>
<td>MCA</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>148 (VPS)</td>
<td>148 (LPS)</td>
</tr>
<tr>
<td>4</td>
<td>50</td>
<td>F</td>
<td>Left</td>
<td>MCA/ACA</td>
<td>2</td>
<td>4</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5</td>
<td>64</td>
<td>F</td>
<td>Left</td>
<td>MCA</td>
<td>2</td>
<td>30</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>70</td>
<td>M</td>
<td>Right</td>
<td>MCA</td>
<td>1</td>
<td>3</td>
<td>58</td>
<td>211</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>55</td>
<td>F</td>
<td>Right</td>
<td>MCA</td>
<td>5</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>8</td>
<td>52</td>
<td>M</td>
<td>Right</td>
<td>MCA/ACA</td>
<td>3</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>99</td>
<td>-</td>
</tr>
<tr>
<td>9</td>
<td>66</td>
<td>M</td>
<td>Right</td>
<td>MCA</td>
<td>1</td>
<td>2</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>10</td>
<td>49</td>
<td>M</td>
<td>Left</td>
<td>MCA</td>
<td>2</td>
<td>5</td>
<td>34</td>
<td>61</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>11</td>
<td>42</td>
<td>F</td>
<td>Right</td>
<td>MCA</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>97 (VPS)</td>
<td>-</td>
</tr>
<tr>
<td>12</td>
<td>72</td>
<td>F</td>
<td>Right</td>
<td>MCA/ACA</td>
<td>8</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>100</td>
<td>-</td>
</tr>
</tbody>
</table>

ACA = anterior cerebral artery, F = female, LPS = lumboperitoneal shunt, M = male, MCA = middle cerebral artery, VPS = ventriculoperitoneal shunt.

Patient 1: a collection was discovered on post-operative day 55 and determined to be secondary to an infection; it was treated with drainage and antibiotics.
signs of hydrocephalus, shunts were not placed. Since these patients remained asymptomatic and did not exhibit was thought to be attributable to cerebral atrophy after the stroke. not shunted also developed slight ventricular enlargement, which were placed for hydrocephalus, respectively. Patients who were lus. Patients 3 and 11 developed ventriculomegaly with ment of a ventriculoperitoneal shunt for underlying hydrocepha- fluid collections. Patient 1, as discussed previously, required place- was performed with a synthetic bone flap.

Patient 10. Neither of these patients had invasive ICP monitoring after surgery, and neither underwent temporal lobectomies (Ta- ble 1). Both received delayed cranioplasties, 211 and 61 days after craniectomy, respectively. Another patient (Patient 1) presented 55 days after surgery with a cranial wound dehiscence, an enlarging collection at the craniectomy site, enlarged ventricles and headaches. The patient was taken to the operating room for wound revision and evacuation of the fluid collection. Cultures from this collection grew methicillin-resistant Staphylococcus aureus and the patient was treated with antibiotics after drainage. As anticipated, the fluid collection resolved immediately after drainage and the patient eventually received a synthetic flap cranioplasty 386 days after her original craniectomy. Given the infectious and delayed appearance of the collection, both relative to the other pa- tients in our study and compared to those in the related literature, we considered this to be a purely infectious collection. The patient also developed hydrocephalus and had placement of a ventriculo- peritoneal shunt.

All surviving patients underwent bone flap replacement or cra- nioplasty for cosmesis and brain protection. No operation was performed with the intention of resolving extra-axial fluid collections. In the entire cohort, the mean time from craniectomy to cranioplasty was 155 days and median time was 119 days (range 61– 386 days). Three patients required placement of shunts (two ventric- ular and one lumbar), none of whom developed extra-axial fluid collections. Patient 1, as discussed previously, required place- ment of a ventriculoperitoneal shunt for underlying hydrocepha- lus. Patients 3 and 11 developed ventriculomegaly with headaches, and ventriculoperitoneal and lumboperitoneal shunts were placed for hydrocephalus, respectively. Patients who were not shunted also developed slight ventricular enlargement, which was thought to be attributable to cerebral atrophy after the stroke. Since these patients remained asymptomatic and did not exhibit signs of hydrocephalus, shunts were not placed.

4. Discussion

Aarabi reviewed a cohort of patients who underwent craniec- tomy following severe head trauma and found that 60% of patients who did not die in the immediate post-operative period developed extra-axial collections or subdural hygromas. Furthermore, these collections occurred ipsilateral to the craniectomy in 92% of cases and most first appeared within one week of surgery. The authors proposed that violation of the dural–arachnoid interface, as inves- tigated by electron microscopy studies,12 is the major physiologic perturbation that gives rise to the collections. These results are supported by the work of our own group (in preparation), in which traumatic patients do present with a high rate of extra-axial fluid collections. This histopathological evidence, combined with the inflammatory cascade that ensues in the setting of trauma, makes these observations biologically plausible. However, our lower rate of collection formation after craniectomy for stroke relative to after head injury suggests that the mechanism for accumulation of fluid collection is a more complex pathophysiologic phenomenon that may be disease-dependent.

Craniectomy surgery violates the dural and bony tissue planes and creates contiguity and communication among the normal cra- nial spaces (subgaleal, epidural, subdural and potentially subarach- noid). Therefore “extra-axial” is the most appropriate anatomical description of the space where post-craniectomy fluid collections occur. The incidence of these collections is also uncertain, with re- ported rates after decompression for MCA stroke ranging from 14% to 64%.1,11 Most importantly, there is no consensus on whether patients with these collections require treatment with shunt or cranioplasty. We evaluated the incidence and behavior of extra-ax- ial collections after decompressive hemicraniectomy for malignant edema after MCA stroke and hypothesize that they represent a dis- tinct clinical entity from subdural hygromas or external hydro- cephalus and that they warrant a different treatment approach.
In this retrospective study, the incidence of extra-axial collection development after decompressive craniectomy for malignant cerebral edema associated with MCA territory infarction was only 18% (two of 11 patients) in the final analysis. We considered a third collection to be attributable to an infected subgaleal collection rather than to a primary perturbation of CSF dynamics; hence this was not defined in the same category as the CSF-based collections that we sought to investigate. The two patients with evidence of extra-axial CSF collections early in the post-operative period (3 and 5 days), demonstrated characteristics consistent with the more typical development of extra-axial collections associated with craniectomy. Importantly, these collections were asymptomatic and resolved spontaneously within 2 months of craniectomy.

Extra-axial fluid collections following decompressive hemicraniectomy have been reported in other studies to be detrimental to recovery and representative of “external hydrocephalus”. It has been suggested that they require treatment with CSF diversion or early cranioplasty to restore normal CSF pressure and dynamics. In a single institution series of decompressive craniectomy for ischemic (n = 11) and hemorrhagic (n = 6) stroke,11 seven of the 11 patients with ischemic stroke developed hydrocephalus, defined as the combination of ventriculomegaly and extra-axial CSF collections or any extra-axial collection. These authors concluded that the extra-axial collections were a result of imbalanced CSF absorption and production and that "external hydrocephalus" was CSF displaced into the extra-ventricular spaces. The collections appeared 7.7 days (mean) after craniectomy (range 1–16 days). A large proportion of their patients received shunts (88%), and the entire cohort underwent cranioplasty at a relatively early mean time of 32 days.

Our data demonstrated that extra-axial CSF collections were observed in only 18% of patients in the acute period, as compared to the 63.3% rate reported by Waziri et al. Furthermore, we observed the spontaneous resolution of the collections in 34 and 58 days, both of which are longer than the average time to cranioplasty in their study. Additionally, our report supports the benign and self-resolving nature of these collections and that their development may be independent of hydrocephalus. It is not immediately clear why one would expect a significant increase in the underlying level of hydrocephalus in stroke patients aside from that developing from post-ischemia atrophy.

Rahme et al. conducted a similar retrospective study to ours, and evaluated 17 patients who underwent decompressive craniectomy for ischemic stroke (n = 12), dural sinus thrombosis, hemorrhagic stroke, and transformation of ischemic to hemorrhagic stroke (combined, n = 5).13 This cohort was similar to ours in the high rate of duraplasty (94.1% compared to 100%), low rate of temporal lobectomy (11.8% compared to 25%) and low rate of ventriculostomy placement (11.8% compared to 8.3%). Rahme et al. also reported that no shunts were necessary for hydrocephalus treatment and they had a low rate of extra-axial collection formation; two patients (14%). Their treatment plan focused on early bone flap replacement and all their patients received cranioplasty within 6 weeks, with a median time from craniectomy to cranioplasty of 21 days. Both of their patients who developed asymptomatic extra-axial collections were treated with prompt cranioplasties, after which the collections resolved. Based on these two results they concluded that early cranioplasty obviated the need for CSF diversion in this patient population. The mean time to cranioplasty in our cohort was 155 days, while it was 21 days and 32 days in previously discussed studies. Although others report resolution of the extra-axial collections after cranioplasty, our data are consistent with spontaneous resolution. This suggests that both CSF shunting and early cranioplasty can be avoided as a treatment for extra-axial collections; a practice that should be beneficial for avoidance of added long-term morbidity.

5. Conclusion

We describe a series of patients who underwent decompressive craniectomy following infarction of the MCA territory. Our goal was to investigate the incidence and management algorithm of extra-axial collections. The low incidence of extra-axial collections after surgery is dissimilar to other series. The asymptomatic nature of the collections and their spontaneous resolution without additional procedures suggests that shunting and early cranioplasty can be avoided in this vexing patient population.

References