Complications of Decompressive Craniectomy

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Resolution of CCFs has been reported after angiography, where a clot developed during the procedure in the internal carotid artery, possibly occluding the arteriovenous connection in a similar mechanism as just described. Similar events have been described soon after gamma knife radiotherapy,7 also potentially secondary to a thromboembolic event from the angiogram used during the treatment planning, and not from an acute radiation effect.

Bujak et al.8 reported 2 patients with dural CCF causing severe clinical manifestations that spontaneously resolved before endovascular intervention. Unlike the present case, obliteration of the CCF was associated with a concomitant resolution of orbital signs and symptoms. Sergot and colleagues5 reported 2 patients with CCF that developed spontaneous thrombosis of the SOV with an acute worsening of symptoms. In contrast to our case, however, thrombosis of the SOV in these 2 patients was not associated with an obliteration of the fistula. One case is therefore unique since, thus far, an acute worsening in the orbital signs and symptoms caused by a spontaneous thrombosis of the SOV and an angiographically documented complete cure of the CCF. Acute thrombosis of the SOV with probable extension proximally into the cavernous sinus accounts for the resolution of the CCF. Since the SOV provides the major and in many cases only venous outflow to the orbit, sudden worsening of orbital congestion manifests as an orbital compartment syndrome (OCS).6 In addition, since the orbital veins are valvesless, some orbital drainage may occur in an autotransfusion fashion from the SOV to the facial venous system and inferiorly through connections with the pterygoidpaleal venousplexus, even with an active CCF. Sudden thrombosis of the SOV may temporarily block off these alternate drainage routes.

Thrombosis of the SOV in all likelihood results in stagnation of abnormal blood flow within the cavernous sinus, precipitating the occlusion of the CCF, slow flow through the clot formation, manifesting as thrombosis. Based on anatomic studies, the SOV is the single major venous drainage for the orbit, resulting in acute orbital, IOP elevation from decreased episcleral venous outflow, and a concomitant optic neuropathy. Once there is no visualization of the CCF on DSA, the endovascular options are limited. Despite the presence of severe orbital signs, the management of the OCS may be difficult. In most cases, the OCS is a transient event, marks- Improving within 48 hours.10 The goal of throbosism of the SOV. Although DSA is the gold standard for diagnosis, there is no role for endovascular therapy and the management is focused on managing the acute orbitalophthalmoplegia and raised intracranial pressure.

Conclusions

Paradoxical worsening of ocular symptoms in presence of complete obliteration of a CCF is extremely rare and possibly triggered by OCS therapy in such situations is to “buy time” until orbital congestion resolves. Presumably, orbital venous outflow forms alternate drainage pathways during this time. Initially, topical anti-glaucoma medications are instituted along with intranasal mirtazapine. If this fails, a lateral canthotomy with cantholysis is performed, but even this may provide only temporary relief, since the OCS will recur as orbital soft tissue congestion fills the decompressed space.

Worsening of the orbital and ocular symp- toms does not always represent persistence or progression of the arterio-venous fistula, as in this case Illustrates. In cases of presumed sponta- neous SOV thrombosis, the use of DSA has been questioned,6 since the diagnosis of SOV thrombosis can be made with MRI. However, the MRI signal characteristic of thrombosis evolve over time and may be difficult to interpret accurately in the SOV. The clinician is then left in a quandary of “waiting out” a possible thrombosis and delaying DSA or proceeding with timely DSA to confirm thrombosis or treat a worsening CCF. Despite the inherent risks of DSA, we support the use of this modality in all cases of acute worsening of orbital signs, since spontaneous SOV thrombosis is a rare event, and delay in definitive care in the face of an acute, severe OCS may result in permanent visual loss.

References

It could also be attributed to high rates of subarachnoid hemorrhage, which has been shown to be associated with increased rates of hydrocephalus.19,34 Waitzi et al. have found a strong correlation between prolonged time to replacement of the bone flap and persistence of hydrocephalus and recommend that early cranioplasty be performed to restore normal intracranial pressure and prevent the development of persistent hydrocephalus.19

Subdural effusion or hygroma
Subdural effusions have been found to be very common after decompressive craniectomy.13,14 The incidence rate across different studies has been found to range from 26% to 60%.13,15 We found that 9% of our patients had subdural hygromas at a mean post-operative day of 16, which was consistent with data from previous studies by Yang et al. and Sivek et al., which reported effusions occurring around 8-30 days post-operation. Studies have attributed the occurrence of subdural effusions to altered CSF dynamics after decompressive craniectomy.13,14 However, many studies show that intervention with hydrogromas are not needed and many resolve on their own. Yang et al. found that 20 out of 23 hygromas resolved on their own without any neurological deficits.34 Arabi et al. and Sivek had similar results.13,14

Herniation
Herniations, defined as brain expansion outside the skull, like subdural hygromas, are a common complication following decompressive craniectomy. They can be a result of hyperperfusion of brain tissue or an increased intracranial pressure due to pinching of cortical veins or laceration of brain tissue near the defect, resulting in ischemia and necrosis of herniated tissue.2 Larger openings have been shown to allow the brain to expand outward with less constriction and can reduce the risk of problems associated with craniotherapy complication.34

Seizures
Our low rates of seizures (1%) could be attributed to the fact that all patients undergoing decompressive craniectomy were placed on an anti-seizure medication, Dilantin (Phenytoin). This was in contrast to Honeybul et al., who found 22% of patients had seizures following decompressive craniectomies, but anti-seizure medication was not used prior to craniotherapy. Ban et al. also used prophylactic antiseizure medication and had lower rates of seizures.19

Syndrome of the Trephined
Syndrome of the trephined, or sinking flap syndrome is characterized by a group of symptoms such as dizziness, seizures, headaches, and mood changes.42 The absence of the bone flap after decompressive craniectomy can cause the scalp to sink into the defect, resulting in the aforementioned symptoms. Early cranioplasty, performed before the flap has sunk in has been recommended, but there has not yet been definitive evidence demonstrating whether this is more beneficial than a later cranioplasty.34,35,36 An alternate procedure known as hinge craniotomy that does not require a subsequent cranioplasty could prevent this syndrome from occurring, and has been suggested to be just as effective as traditional cranioplasty.36-46

Parameters affecting cranioplasty outcomes
The literature has demonstrated two major methods for preserving the bone flaps after decompressive craniectomy, either in the freezer or subcutaneously.1,34,35,11,13,47 In addition, there has been a method described where the bone flap is replaced as part of the procedure and connected to the rest of the skull in a hinged fashion.48 There have been limited studies looking at the complications of this method compared to traditional craniotherapy after decompressive craniectomy. Of the studies that did, both demonstrated that hinged craniotomy was just as effective as decompressive craniectomy and eliminated the need for a cranioplasty procedure.35,48 In this study, we looked at infection rates following cranioplasty and differences in bone flap preservation across multiple studies (Table 4). Our infection rate (21%) was higher than other studies. This could be attributed to our method of storing bone flaps in the freezer, in addition to the high rate of synthetic bone flap use, which has been shown to be associated with higher rates of infection.15

Discussion
Brain edema requiring medical intervention occurs in a variety of conditions and may cause ICP elevation. Persistent ICP elevations have been associated with poor clinical outcomes after aneurysm rupture.34,35 Decompressive craniectomy is a relatively quick surgical procedure that is able to relieve elevating pressures. However, despite many studies demonstrating its efficacy in reducing ICP, there remain questions about the complications following DCRs and whether certain preoperative parameters can better predict the chances of developing complications.11,12 Despite many studies looking into the efficacy of the procedure, limited studies have attempted to look at the complications following decompressive craniectomies and its association to infection that required bone flap removal. Eleven patients used a synthetic bone flap made of either titanium mesh or methylmethacrylate. Infection requiring bone flap removal occurred in 9 (11%) of our patients, with a mean infection time of 156 days and ranged from 11-540 days. Table 2 shows the data of the patient population who underwent craniotherapy after decompressive craniectomy.

Complications
Complications such as herniation, subdural effusion, seizures, hydrocephalus, hematoma and infection have been found to occur across different studies. The fluctuation in the rates between the studies may indicate differences in procedure protocols, differences in time between incising injury and the Table 3. Cranioplasty Patient Characteristics

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<tr>
<td>Total no. cranioplasties</td>
<td>90</td>
<td>90</td>
<td>108</td>
<td>138</td>
<td>35</td>
<td>62</td>
<td>53</td>
</tr>
<tr>
<td>Autologous</td>
<td>62 (69%)</td>
<td>Not used</td>
<td>33 (94%)</td>
<td>57 (92%)</td>
<td>42 (79%)</td>
<td>42 (79%)</td>
<td>89</td>
</tr>
<tr>
<td>Synthetic</td>
<td>11 (12%)</td>
<td>80</td>
<td>9 (9%)</td>
<td>4 (8%)</td>
<td>3 (6%)</td>
<td>2 (6%)</td>
<td>28</td>
</tr>
<tr>
<td>Range (days)</td>
<td>3-1200</td>
<td>3-1200</td>
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<td>3-1200</td>
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<td>3-1200</td>
<td>3-1200</td>
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<tr>
<td>Bone flap storage length</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
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Table 4. Complication Details in Multiple Studies
A short time between craniectomy and cranio- 
plasty has been associated with poorer outcome.

...found that cranio- 
plasties taking place 1-6 months after craniectomy had the highest complication rate (79%) compared to those performed 12-18 months after craniectomy (4.5%). However, Beaufraich et al. found that cranio- 
plasties taking place 2-6 weeks, rather than the more typical 3-6 months did not produce significantly more complications. They also found that there were higher rates of infection in patients that were not properly medicated with antibiotics.

...a higher rate of infection in patients that had their bone flaps stored in a freezer com- 
parison to those stored at room temperature. The differences in infection rates may be attributed to limited follow-up. As a result of limited 
follow-up, no measure of long-term outcomes were made. We used GCS as an outcome mea- 
surement, which could be argued to be a fairly vague neurological assessment. The differences in time between craniectomy and cranio- 
plasty were due to inter-surgeon variations on the best time to perform a craniectomy. The vast majority of patients in this study were SAH patients, with very few TBI patients. There may be differences in the outcome of decompressive craniectomy in SAH versus TBI patients.

Conclusions

Decompressive craniectomy is a proven method used to reduce intracranial pressure. However, there are numerous complica- 
tions associated with this procedure. This study, unlike many prior studies that included patients with traumatic brain injury, mainly had patients that suffered from subarachnoid hemorrhage. Also, unlike the other studies that found associations between preoperative GCS scores, age and the development of complica- 
tions, our study did not find any significant associations between age, gender, diagnosis and preoperative GCS score with the incidence or total number of complications. Such results argue against the possibility of potential predi- cators of complications in patients that suffer from subarachnoid hemorrhage and suggest that predictors of complications may depend on the type of injury.

There was no association between age and death from decompressive craniectomy. Older patients generally tended to have better GCS scores upon discharge, but female patients and patients with any complication tended to have lower GCS discharge scores.

In comparing our data along with the other studies utilizing freezer storage with studies utilizing subarachnoid bone flap storage there was a higher rate of infection in patients that had their bone flaps stored in a freezer com- 
parison to those stored at room temperature. The differences in infection rates may be attributed to limited follow-up. As a result of limited 
follow-up, no measure of long-term outcomes were made. We used GCS as an outcome mea- 
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