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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, 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.3,4 reported 2 patients with 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 colleagues7 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 there was 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 SOV with probable extension proximally into the cavernous sinus accounts for the reso- lution of the CCF. Since the SOV provides the major drainage of the anterior cranial venous system for the orbit, sudden worsening of orbital congestion manifestations as an orbital compartment syndrome (OCS).5 In addition, since the orbital veins are valveless, some orbital drainage may occur in an antegrade fashion from the SOV to the facial venous system and inferorly through connections with the pterygopatine venousplexus, even with an acute CCF. Sudden thrombosis of the SOV may temporarily block off these alternative draining 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 in the coagulum cascade, manifesting as thrombosis. Based on anatomic studies, the SOV in this particular case was 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, markedly improving within 48 hours.6 The goal of 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 intravenous nimodipine. 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 symptoms does not always represent persistence or progression of the arterio-venous fistula, as in this case Illustrates. In cases of presumed spontaaneous SOV thrombosis, the use of DSA has been questioned,8 since the diagnosis of SOV thrombosis can be made with MRI. However, the MR 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.

Conclusions
Paradoxical worsening ofocular symptoms in presence of complete obliteration of a CCF is extremely rare and possibly triggered by thrombosis of the SOV. Although DSA is the gold standard for diagnosis, there is no role for endovascular intervention and the management is focused on managing the acute orbitalopathy and raised intracranial pressure.

References

Figure 4
MRI Gradient Echo sequence showing (arrow) a hypointense SOV compatible with thrombosis within

Table 1. Complications following Decompressive Craniectomy

<table>
<thead>
<tr>
<th>Complication</th>
<th>N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydrocephalus</td>
<td>3 (2.1)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>17 (12.4)</td>
</tr>
<tr>
<td>Herniation</td>
<td>40 (29.3)</td>
</tr>
<tr>
<td>Vasospasm</td>
<td>10 (7.2)</td>
</tr>
<tr>
<td>Subdural hygroma</td>
<td>18 (12.9)</td>
</tr>
<tr>
<td>Seizures</td>
<td>2 (1)</td>
</tr>
<tr>
<td>Sphenoid flap</td>
<td>2 (1)</td>
</tr>
<tr>
<td>Flap revision</td>
<td>0</td>
</tr>
<tr>
<td>Increased ICP</td>
<td>(4.7)</td>
</tr>
<tr>
<td>Intracranial hypertension</td>
<td>21 (15.8)</td>
</tr>
<tr>
<td>*Pneumonia was the commonest infection in this study</td>
<td></td>
</tr>
</tbody>
</table>

Note: This study was supported by grants from the National Institute of Neurological Disorders and Stroke.
It could also be attributed to high rates of subarachnoid hemorrhage, which has been shown to be associated with increased rates of hydrocephalus.18-21 Wartsi 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.18

Subdural effusion or hydrogroma
Subdural effusions have been found to be very common after decompressive craniectomy.11-13 The incidence rate across different studies has been found to range from 26% to 60%.11-13 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 Siwek 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.11-13 However, many studies show that interventions with hygromas 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 neurologically deficits.11,13 Arah et al. and Siwek et al. have had similar results.11-13

Herniation
Herniations, defined as brain expansion outside the skull, such as subdural hygromas, represent a decompressive craniectomy complication. They can be a result of hyperperfusion of brain tissue or an increased transcapillary leakage due to the drop in intracranial pressure.11,13 This can cause shearing of cortical veins or laceration of brain tissue near the defect opening, resulting in ischemia and necrosis of herniated tissue.11 Lacerations have been shown to allow the brain to expand outward with less constriction and can reduce the risk of problems associated with the decompressive craniectomy.11,13

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 Honeybol et al., who found 22% of patients had seizures following decompressive craniectomies, but anti-seizure medication was not given until cranioplasty, unless the patient was already on such medication.14 Ban et al. also used prophylactic antiepileptic medication and had lower rates of seizures.14

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.1,12 The absence of the bone flap after decompressive craniectomy can cause the scalp to sink into the defect, resulting in the aforementioned symptoms. Early craniectomy, 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 craniectomy.11,13-15 An alternate procedure known as hinge cranioplasty that does not require a subsequent cranioplasty could prevent this syndrome from occurring, and has been suggested to be just as efficacious as decompressive craniectomy.11,13

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.11,13,14,16 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 hinge fashion.11 There have been limited studies looking at the complications of this method compared to traditional cranioplasty after decompressive craniectomy. Of the studies that did, both demonstrated that hinge cranioplasty was just as effective as decompressive craniectomy and eliminated the need for a cranioplasty procedure.39,40 In this study, we looked at infection rates following cranioplasty and differences in bone flap preservation across multiple studies (Table 4). Our infection rate (2%) 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.11
A short time between craniotomy and cranioplasty has been associated with poorer outcomes40-43. Ribot et al. found that cranioplasties taking place 1-6 months after craniotomy had the highest complication rate (79%) compared to those performed 12-18 months after craniotomy (4.5%).44 However, Beauchamp et al. found that earlier cranioplasties 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 had their bone flap stored in a freezer compared to those that were stored on wet towels.11 Certainly, larger scale prospective studies are warranted to determine the risk and benefits of both bone flap storage methods.

References