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Complications of Decompressive Craniectomy

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patients with CCF that developed spontaneous edly improving within 48 hours.10 The goal of management of the OCS may be difficult. In DSA, the endovascular options are limited.

the orbit, resulting in acute orbitopathy, IOP through connections with the pterygopalatine may occur in an anterograde fashion from the cavernous sinus accounted for the resolu-

tion of the CCF. Since the SOV provides the major and in many cases only venous outflow with probable extension proximally into the SOV and an angiographically documented thrombosis can be made with MRI. However, in presence of complete obliteration of a CCF spontaneous SOV thrombosis, the use of DSA has been questioned,10 since the diagnosis of SOV thrombosis and delaying DSA or proceeding until orbital congestion resolves. Presumably, orbital venous outflow forms alternate drain-

age pathways during this time. Initially, topical anti-glucoma 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 recoc as orbital soft tissue congestion fills the decompressed space. Worsening of the orbital and ocular sympto-

ms does not always represent persistence or progression of the artery-venous fistula, as in this case Illustrates. In cases of presumed sponta-

neous SOV thrombosis, the use of DSA has been questioned,10 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 distin-

guish 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.

Conclusion

Paradoxical worsening of ocular symptoms in presence of complete obliteration of a CCF is extremely rare and possibly triggered by 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 orbitalopathy and raised intracranial pressure.

References


Introduction

Persistent elevation of intracranial pressure (ICP), if untreated, may lead to brain ischemia or lack of brain oxygen and even brain death.1-6,10 When standard treatments for elevated ICP are exhausted without any signs of improvement, decompressive craniectomy can be an effective alternative solution.7,8,10

Decompressive craniectomies (DC) have been used as a method of controlling intracranial pressure in patients with cerebral edema secondary to cerebral ischemia, subarachnoid hemorrhage (SAH), and traumatic brain injury (TBI), among others.11 Several studies over the years have demonstrated the efficacy of this procedure.7,9,10,11 However, consensus is still lacking in the utility of DC as an effective first tier treatment for intratable intracranial pressure due to the rudimentary neurological outcome assessments, and the many complications associated with this procedure.11,12,13

There are a limited number of studies that have looked at complications secondary to the procedure itself.9,10 The majority of these studies only investigated the impact of this procedure in patients with traumatic brain injury. The purpose of this study is to investigate the rates of various complications associated with the decompressive craniectomy procedure in patients that did not suffer from traumatic brain injury, and to determine whether the same associations between preoperative parameters and development of complications can be made.

Methods

A retrospective review of a prospectively collected data set of patients who had a decompressive craniectomy done at our institution between January 2003 and January 2010 was performed. Electronic charts were reviewed to obtain the following data: patient age, gender, diagnosis, type of decompressive craniectomy, any complications following the procedure, patient outcome as measured by Glasgow coma scale (GCS) at discharge, time period between craniectomy and cran
tomy and type of flap used for cranioplasty. Rates of various complications were tabulated and we investigated the association of several patient parameters with patient outcome, and rates of the various complications. These factors included age, gender and preoperative GCS.

Appropriate statistical tests were used to determine the strength of associations. Spearman’s r, Student’s t-test and multivariate regression were performed using the JMP statistical package (version 7.02; SAS Institute, Cary NC).

Results

191 patients were identified, including 99 females, 91 males. The mean age was 50 years old (range 17-85). The mean preoperative GCS score was 8 (range 3-15). 70 patients had intracerebral hemorrhage (48.6%), 60 had ruptured aneurysm (31.4%), 21 had brain edema secondary to a prior elective brain surgery (11.5%), 15 had stroke (7.8%), 11 had closed head trauma (5.7%), 4 had thrombosed aneurysm (2.1%), 3 had ruptured arteriovenous malformation (AVM) (1.6%), 2 had penetrating trauma (1.1%), 1 had tumor (0.5%), and 3 were unreported (1.6%). A bifrontal craniectomy was performed on 4 cases (2.1%) and 187 were unilateral craniectomies (97.9%). 40 patients (21.1%) had trauma (1.4%), 1 had tumor (0.5%), and 3 were unreported (1.6%). A bifrontal craniectomy was performed on 4 cases (2.1%) and 187 were unilateral craniectomies (97.9%). The incidences of paralysis were questioned,10 since the diagnosis of SOV 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 of ocular symptoms in presence of complete obliteration of a CCF is extremely rare and possibly triggered by 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 manner as just described. Similar events have been described soon after gamma knife radiotherapy,2 also potentially secondary to a thromboembolic event from the angiomat from the treatment planning, and not from an acute radiation effect.

Bux et al.3 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 colleagues4 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. Our case is therefore unique, since there was an acute worsening in the orbital signs and symp- toms 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 accounted for the resolu-

tion of the CCF. Since the SOV provides the major and in many cases only venous outflow,
fewer number of complications had a higher GCS score upon discharge (Spearman’s rho = -0.1717, p=0.064).

As a priori analysis comparing various patient parameters (age, gender, diagnosis, initial GCS and delta GCS) against rate of the various complications per patient did not reveal any statistically significant association. Cranioplasty was performed in 90 patients, with 19 patients needing to undergo reoperation due to infection that required bone flap removal. In 62 patients, autologous bone flap was used. Eleven patients used a synthetic bone flap made of either titanium mesh or methylmethacrylate. The average time between craniectomy and cranioplasty was 156 days and ranged from 51-540 days. Table 2 shows the data of the patient population who underwent cranioplasty after decompressive craniectomy.

### 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. 13, 14 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 followed.19-27 And whether certain preoperative parameters can better predict the chances of developing complications.”

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 with preoperative measurements such as age, gender and preoperative GCS score.19-27 Table 3 summarizes the complications from different studies. Among the studies, most common complications were subdural effusion and hydrocephalus. 15, 16, 17, 18, 19 Unlike prior studies that included only patients with traumatic brain injury, our study consists mainly of patients that suffered from subarachnoid hemorrhage.

### Complications

Complications such as herniation, subdural effusion, seizures, hydrocephalus, hematoma and infection have been found consistently in various studies. The fluctuation in the rates between the studies may indicate differences in diagnosis and classification, differences in time between inciting injury and the decompressive craniectomy procedure, average age of patients or type of injury. Ban et al. have found that age (66) and a Glasgow Coma Scale (GCS) of less than 8 related to the development of complications. 13 Stiver et al and have also reported that patients with lower preoperative GCS score and age greater than 50 years had a higher risk of developing a complication.19 Cooper et al, in a recent randomized prospective controlled Decompressive Craniectomy (DECRa) trial, found that those assigned to have a decompressive craniectomy procedure, 3% developed one or more complications, compared to the standard-care group with 17%.30, 31 Our analysis revealed no statistically significant associations between patient parameters such as age, gender and initial GCS with the rates of individual complications or the total number of complications in a single patient. Such results argue against the possibility of potential predictors of complications in patients undergoing decompressive craniectomy. It is worth noting that the three aforementioned studies included primarily traumatic brain injury patients, unlike our study, which may account for the differences in the results.

### Hydrocephalus

The incidence of hydrocephalus following decompressive craniectomy ranges from 10% to 40%, 15, 26, 27, 30. Our rates of hydrocephalus were high compared to other studies, but this could be due to inconsistencies in diagnostic criteria as described in previous studies. 27, 28

It could also be attributed to high rates of subarachnoid hemorrhage, which has been shown to be associated with increased rates of hydrocephalus. 29, 30 Watan 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. 31 Subdural effusion or hygroma Subdural effusions have been found to be very common after decompressive craniectomy. 9, 12, 14 The incidence rate across different studies has been found to range from 26% to 80%. 32, 33, 34, 35 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 Stiver 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. 35, 36 However, many studies show that intervention 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 neurological deficits. 37, 38 Arani et al and Stiver had similar results. 9, 13, 12

### 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 a hyperextension of brain tissue or an increased transcranial leakage due to the drop in intracranial hydrostatic pressure. 9 This can cause stretching of cortical veins or laceration of brain tissue near the defect opening, resulting in ischaemia and necrosis of herniated tissue. 39 Larger openings have been shown to allow the brain to expand outward with less constriction and can reduce the risk of problems associated with cranioplasty. 14

### Seizures

Our low rates of seizures (1%) could be attributed to the fact that all patients undergoing decompressive craniectomies 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 initiated until 7 days after cranioplasty, unless the patient was already on such medication. 40, 41 Ban et al. also used prophylactic anti-seizure medication and had lower rates of seizures. 42

### 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. 43, 44 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 can be recommended, but there has not yet been definitive evidence demonstrating whether this is more beneficial than a later cranioplasty. 45, 46 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 traditional cranioplasty. 

### 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. 47, 48, 49, 50, 51, 52, 53 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. 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. 51, 52

In this study, we looked at infection rates following cranioplasty and differences in bone flap preservation across multiple studies (Table 4). Our infection rates (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.
A short time between craniectomy and cranioplasty has been associated with poorer outcome.

Roth et al. found that cranioplastic skull replacement lagging 1-6 months after craniectomy had the highest complication rate (79%) compared to those performed 12-18 months thereafter (4.5%).

However, little research has been done on whether the time between craniectomy and cranioplasty influences the rate of complications. Certainly, large, scale prospective studies are warranted to determine the risk and benefits of both bone flap storage methods.

References

Limitations
There was no randomization in this study. Most of the patients used in this study did not suffer from severe brain injury. The low incidence of cranioplasty and bone flap resorption may have led to underestimation of complications.

Conclusions
Decompressive craniectomy is a proven method used to reduce intracranial pressure. However, many studies have reported that the complications 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 progressive GCS scores, age and the development of complications, our study did not find any significant associations between age, gender, diagnosis and postoperative GCS score with the incidence or total number of complications. Such results argue against the possibility of potential predictors 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 deaths from decompressive craniectomy. Older patients generally tend to have better GCS scores upon discharge, but female patients had more deaths with patients with any complication tended to have lower GCS discharge scores.

In comparing our data along with the other studies utilizing lower GCS scores for bone flap storage there was a higher rate of infection in patients that had their bone flaps stored in a freezer compared to those that were stored at 4°C. Cerebrum measurement techniques were used to determine the risk and benefits of both bone flap storage methods.

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