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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,[9] possibly occluding the arteriovenous connection in a similar mechanism as just described. Similar events have been described soon after gamma knife radiotherapy,[8] 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[4] 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. Sergott and colleagues[10] 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 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 accounted for the resolution of the CCF. Since the SOV provides the major and in many cases only venous outflow for the orbit, sudden worsening of orbital congestion manifests as an orbital compartment syndrome (OCS).[2] In addition, since the orbital veins are valveless, some orbital drainage may occur in an anterograde fashion from the SOV to the facial venous system and inferiorly through connections with the pterygopalatine venous plexus, 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 triggers the coagulation 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 orbitopathy, IOP elevation from decreased episcleral venous outflow, and a congestive 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.[10] 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 mannitol. 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.

Figure 4

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

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 spontaneous 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[6] and may be difficult to interpret accurately in the SOV. The clinician is then left in a quandry 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 of ocular 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 therapy and the management is focused on managing the acute orbitopathy and raised intraocular pressure.

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Complications of Decompressive Craniectomy

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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.[7,19]

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. [8-10] Several studies over the years have demonstrated the efficacy of this procedure.[7-9,11,35,36] However, consensus is still lacking in the utility of DC as an effective first tier treatment for intractable intracranial pressure due to the rudimentary neurological outcome assessments, and the many complications associated with this procedure.[11,12,59]

There are a limited number of studies that have looked at complications secondary to the procedure itself.[13-18] 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 cranioplasty 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 p, 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 (36.6%), 60 had ruptured aneurysm (31.4%), 21 had brain edema secondary to a prior elective brain surgery (11%), 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 (1.4%), 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 complications are summarized in Table 1.

101 of the 191 patients (53%) had at least one complication. 42 patients died despite the procedure. Of the survivors (n = 149), a significant number were discharged to rehabilitation (n=121), 8 were discharged to full time nursing facilities, 2 remained in the hospital, 1 was discharged to hospice, and the rest returned home (n = 13). Three cases did not report discharge destination. There was no correlation between age and mortality.

19 patients had a preoperative GCS score ranging from[3-5, 49] patients ranged from 6-9 and 33 patients were greater than 9. The mean preoperative score was 8. Twelve patients had a postoperative GCS score of 6 or less, 40 were between 6-9 and 68 patients had scores greater than 9. Mean postoperative GCS scores were 3.87±0.49 (mean±SE) above preoperative GCS scores. Patients with higher pre-op GCS scores or older age tended to have higher GCS upon discharge ($p<0.091$). Female patients and patients that had one or more complications had lower GCS scores upon discharge ($p<0.037, p<0.016$). Neither gender nor age was associated with either incidence or total number of complications. Patients that had a

Table 1. Complications following Decompressive Craniectomy

Complication	N (%)
Hydrocephalus	55 (28.7)
VP shunt	37 (19.4)
Herniation	40 (20.9)
Vasospasm	10 (5.2)
Subdural hygroma	18 (9.4)
Seizures	2 (1)
Sunken flap	2 (1)
Flap resorption	0
Increased ICP	9 (4.7)
Infection*	42 (21.9)

*Pneumonia was the commonest infection in this study

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fewer number of complications had a higher GCS score upon discharge (Spearman's rho = -0.1717, p=0.064).

An a priori analysis comparing various patient parameters (age, gender, diagnosis, initial GCS and delta GCS) against rate of the various individual complications and total number of 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 11-540 days. Table 2 shows the data of the patient population who underwent cranioplasty after decompressive craniectomy.

Table 2. Cranioplasty Patient Characteristics

	N (%)
Total cranioplasty procedures	90
Autologous flapt	62 (69%)
Synthetic flapt	11 (12%)
Not recorded	17 (19%)
Average time between craniectomy and cranioplasty (days)	156
Range (days)	11-540
Infection requiring bone flap removal	19 (21%)
Autologous flap infection	11 (18%)
Synthetic flap infection	8 (73%)
Bone flap resorption	0 (0%)

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.[29-32] 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 DCs and whether certain preoperative parameters can better predict the chances of developing complications.[19-27]

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.[13-15] Table 3 summarizes the complications from different studies. Among the studies, two of the most common complications were subdural effusions and hydrocephalus.[13,14,16,17,19,33] Unlike prior studies that included mostly 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 across different studies. The fluctuation in the rates between the studies may indicate differences in procedure protocols, 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 (≥65) and a Glasgow Coma Scale (GCS) of less than 8 related to the development of complications.[16] Stiver has also reported that patients with lower preoperative GCS score and greater age had a higher risk of developing a complication.[15] Cooper et al., in a recent randomized prospective controlled Decompressive Craniectomy (DECRA) trial, found that of those assigned to have a decompressive craniectomy procedure, 37% developed one or more complications, compared to the standard-care group with 17%.[59]

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%.[15,35,51-55] 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.[7,17,57] It could also be attributed to high rates of subarachnoid hemorrhage, which has been shown to be associated with increased rates of hydrocephalus.[60,61] Waziri 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.[59]

Subdural effusion or hygroma

Subdural effusions have been found to be very common after decompressive craniectomy.[13,48] The incidence rate across different studies has been found to range from 26% to 60%.[15,17,19] 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.[53-55] 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.[17] Arabi et al. and Stiver had similar results.[15,19]

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 transcapillary leakage due to the drop in interstitial hydrostatic pressure.[15] This can cause pinching of cortical veins or laceration of brain tissue near the defect opening, resulting in ischemia and necrosis of herniated tissue.[7] Larger openings have been shown to allow the brain to expand outward with less constriction and can reduce the risk of problems associated with this complication.[3]

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 Honeybul et al., who found 22% of patients had seizures following decompressive craniectomies, but anti-seizure medication was not used prior to cranioplasty, unless the patient was already on such medication.[13] Ban et al. also used prophylatic antiepileptic medication and had lower rates of seizures.[16]

Table 3. Literature Summary of Complications following Decompressive Craniectomy

Complication	This Study	Ban et al (2010)	Yang et al (2008)	Honeybul (2010)	Honeybul et al (2010)	Huang et al (2008)	Aarabi et al (2006)
Herniation	40 (21%)		30 (28%)	21 (51%)	43 (26%)		
Subdural effusion	17 (9%)	29 (33%)	23 (21%)	25 (62%)	80 (49%)	10 (26%)	25 (50%)
Seizures	4 (1%)	4 (4%)	3 (3%)	6 (14%)	36 (22%)		
Hydrocephalus	55 (29%)	10 (11%)	10 (9%)	5 (11%)	23 (14%)	3 (8%)	5 (10%)
Bone flap infection	27 (14%)			5 (11%)	20 (12%)		2 (6%)
Hematoma	17 (9%)	5 (6%)	8 (7%)			2 (5%)	
Infection*	36 (19%)	4 (5%)	3 (3%)			2 (5%)	1 (2%)
Bone flap resorption	0			7 (17%)	11(7%)		6 (12%)
Total no. patients	191	89	108	41	164	38	50
Mean age	50	51	44	32		43	25

*Includes Pneumonia, urinary tract infection, sepsis, and staphylococcus infection.

Table 4. Cranioplasty Details in Multiple Studies

Complication	This Study	Stephens et al	Honeybul et al	Honeybul	Gooch et al	Movassaghi et al	Shoakazemi et al	Tybor et al
Total no. cranioplasties	90	108	138	35	62	53	89	28
Autologous	62 (69%)	Not used		33 (94%)	57 (92%)	42 (79%)	89	28
Synthetic	11 (12%)	108		2 (6%)	5 (8%)	8 (15%)		
Time between craniectomy & cranioplasty (days, avg.)	156	190	94	87	129	95	42	14
Range (days)	11-540	0-360+	44-127	25-274	0-137+	15-388	8-305	8-53
Infection requiring bone flap removal	19 (21%)	9 (8%)	16 (12%)	4 (11%)	7 (11%)	3 (6%)	5 (6%)	1 (4%)
Autologous infection	11			3	0	3	5	1
Synthetic infection	8	9		1	0	0		
Bone flap resorption	0 (0%)		14 (10%)	6 (17%)	4 (7%)		2 (2%)	
Bone flap storage location prior to cranioplasty	Freezer	Synthetic (-40°)	Freezer (-35°)	Tissue Bank	Subcutaneous Storage	Subcutaneous storage	Subcutaneous storage	

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.[50] 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 has been recommended, but there has not yet been definitive evidence demonstrating whether this is more beneficial than a later cranioplasty.[42-45,50] 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 efficacious as decompressive craniectomies.[49,40]

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.[13,14,34,36-38] 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. 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 craniotomy 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 (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.[45]

A short time between craniectomy and cranioplasty has been associated with poorer outcome43-45. Rish et al. found that cranioplasties taking place 1-6 months after craniectomy had the highest complication rate (79%) compared to those performed 12-18 months after craniectomy (4.5%)41. However, Beauchamp et. al found that earlier cranioplasties taking place at 2-6 weeks, rather than the more traditional 3-6 months did not produce significantly more complications. They also found that there were higher rates of infection in those that used synthetic materials compared with those that had autografts45.

Limitations

There was no randomization in this study. Most of the patients used in this study did not suffer from traumatic brain injury. The low incidence of bone flap resorption 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 measurement, which could be argued to be a fairly vague neurological assessment. The differences in time between craniectomy and cranioplasty were due to inter-surgeon variations on the best time to perform a cranioplasty. 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 intractable intracranial pressure. However, there remain numerous 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 preoperative GCS scores, age and the development of complications, our study did not find any significant association between age, gender, diagnosis and preoperative 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 also 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 subcutaneous 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 subcutaneously. Certainly, larger scale prospective studies are warranted to determine the risk and benefits of both bone flap storage methods.

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