Bifrontal decompressive craniotomy for malignant brain edema

Sherif Elwatidy, *FRCS(SN), MD.*

ABSTRACT

Objective: To review the outcome of bifrontal decompressive craniotomy used for the treatment of malignant brain edema due to different etiologies.

Methods: The study was carried out at King Khalid University Hospital, Riyadh, Kingdom of Saudi Arabia during the period from January 2000 to June 2005, and included all patients who had malignant brain edema due to different etiology and were treated with bifrontal decompressive craniotomy after failure of aggressive medical treatment.

Results: Ten patients were included in the study, 6 males and 4 females; the mean age was 24 years. Seven patients had severe head injury, 2 had aneurysmal subarachnoid hemorrhage, and one had large calcified olfactory groove meningioma. Clinically, all patients, except one, had Glasgow coma scores more than 3 before surgery, and operation was performed in all patients once clinical deterioration was observed and diagnosis confirmed by CT brain scan. The outcome of surgery was good in 70%, poor in 20%, and mortality was 10%. The mean hospital stay was 85 days.

Conclusion: Bifrontal decompressive craniotomy offers immediate reduction of intracranial pressure to its normal levels, and improves the outcome of malignant brain edema whatever its cause, it should be performed once clinical deterioration is observed.

Neurosciences 2006; Vol. 11 (4): 241-247

Malignant brain edema is a situation of severe, progressive and diffuse cerebral edema that causes rapid clinical deterioration, which does not respond to aggressive treatment. It is usually seen in patients with; type III severe head injury (SHI), aneurysmal subarachnoid hemorrhage (SAH), and massive brain infarction. Clinically, malignant brain edema is manifested as herniation syndrome in the form of rapid deterioration of consciousness associated with posturing and pupillary changes (mydriasis, anisocoria, and loss of light reflex). Radiologically, there is compression of the ventricles, obliteration of the basal cisterns, loss of normal gyral pattern, and poor grey-white matter differentiation. Despite the advances in understanding, monitoring, and treating cerebral hypertension, the outcome of patients with refractory intracranial hypertension (IH) remains poor; 34% mortality, 40% severe disability including vegetative patients, 13% moderate disability, and 3% good outcome.[1,2] The concept of wide bone removal for treatment of IH has been recognized since the nineteenth century; different types of decompressive craniectomy have been described including unilateral or bilateral frontal and subtemporal decompression, and circumferential hemicraniectomy.[3-7] Bifrontal decompressive craniotomy (BDC) was initially described by Miyazaki in 1966,[7] and was popularized by Kjellberg and Prieto in 1971.[8] The American

From the Division of Neurosurgery, King Khalid University Hospital, College of Medicine, King Saud University, Riyadh, *Kingdom of Saudi Arabia*.

Received 21st December 2005. Accepted for publication in final form 9th April 2006.

Address correspondence and reprint request to: Dr. Sherif Elwatidy, Associate Professor & Consultant Neurosurgeon, Division of Neurosurgery, King Khalid University Hospital, College of Medicine, King Saud University, PO Box 7805, Riyadh 11472, *Kingdom of Saudi Arabia*. Tel. +966 (1) 4671575. Fax. +966 (1) 4679493. E-mail: smfwat@yahoo.com

Association of Neurological Surgeons[9] has recommended decompressive craniotomy for patients with traumatic brain injury (TBI) and refractory IH if some or all of the following criteria were met: 1. Diffuse cerebral swelling on cranial CT imaging, 2. Within 48 hours of injury, 3. No episodes of sustained intracranial pressure (ICP) >40 mm Hg before surgery, 4. Glasgow coma score (GCS) >3 at some point subsequent to injury, 5. Secondary clinical deterioration, and 6. Evolving cerebral herniation syndrome.

The main objective of the study is to review the outcome of patients who had surgery in the form of BDC for treatment of malignant brain edema due to different etiologies after failure of aggressive medical treatment, and to shed light on its immediate and sustained control of raised ICP.

Methods. The study was carried out at King Khalid University Hospital, Riyadh, Kingdom of Saudi Arabia during the period from January 2000 to June 2005; it included patients who developed intractable brain edema regardless of the etiology. Patients were managed in the intensive care unit (ICU) according to standardized protocol which included: CT brain scan as early as possible after resuscitation, placement of ICP monitor for patients with GCS less than 10, hyperventilation to maintain PCO_2 at 30-35 mm Hg, mild head elevation, and mannitol. If the previous measures fail to control the ICP, ventriculostomy with CSF drainage, barbiturate coma, indomethacin, and hypothermia were used as second line therapy. When all measures failed, patients were taken to theater for BDC. The surgical technique of BDC is basically a modification of the technique described by Kjellberg and Prieto in 1971.[10] The medical records of patients were reviewed; patient's age, sex, pathology, GCS on admission and after deterioration were recorded. Radiological data included CT scan findings on admission, following deterioration, and at follow up. The timing of surgery, postoperative complications, and timing of bone flap replacement were included in the data sheet. Glasgow outcome score (GOS) at the time of discharge, total duration of hospital stay (determined by patient's death or discharge from hospital), follow up period, and the final outcome was documented.

Results. The study included 10 patients, 6 males and 4 females; the patient's age ranged from 6 months to 60 years (mean 24 years). The etiology of intractable brain edema was traumatic in 7, and non traumatic in 3 patients (aneurysmal SAH in 2 patients, and excision of large olfactory groove meningioma in the third one). All patients had GCS score above 3 before surgery. **Table 1** summarizes the clinical and radiological findings of the patients. The timing of surgery was determined by the observed clinical deterioration and the radiological findings (compression of the lateral ventricles and obliteration of the basal cisterns), as well as the raised ICP (>30 mm Hg for 30 minutes). Postoperative complications included wound infection in one patient, bone flap resorption in one patient, and hydrocephalus that required ventriculo-peritoneal shunt in 3 patients. The total hospital stay for survivors ranged from 40-140 days (mean 85), and the follow up period ranged from 6 months to 4 years (mean 18 months). Clinical outcome was assessed at a mean of 12 months postoperatively, 7 patients (70%) had favorable outcome (GOS 5 and 4), 2 patients (20%) had poor outcome but were not vegetative, and one patient (10%) died, his GCS score was 3/15 on admission and both pupils were dilated and fixed.

Patient 1. A 6-year-old boy who was admitted to hospital following a road traffic accident; he sustained

Table 1 – Clinical and radiological findings of patients included in the study.

Age	Gender	Pathology	GCS changes	Days	Complications	Outcome
16 years	M	SHI	7 - 4	60 days	None	Good
6 months	F	SHI	10 - 6	90 days	Hydrocephalus required V-P shunt	Fair
18 years	M	SHI	8 - 5	80 days	Bone resorption	Good
6 years	M	SHI	6 - 4	60 days	None	Good
30 years	F	Meningioma	15 - 5	120 days	None	Good
60 years	F	SAH	14 - 4	90 days	Chest infection and deep vein thrombosis	Fair
55 years	F	SAH	14 - 5	140 days	Wound infection, hydrocephalus required V-P shunt	Poor
25 years	M	SHI	7 - 5	40 days	Hydrocephalus required V-P shunt	Good
52 years	M	SHI	10 - 5	5 days	Massive brain infarction	Died
6 years	M	SHI	7 - 5	90 days	Chest infection and urinary tract infection	Good

SHI – severe head injury, SAH – aneurysmal subarachnoid hemorrhage

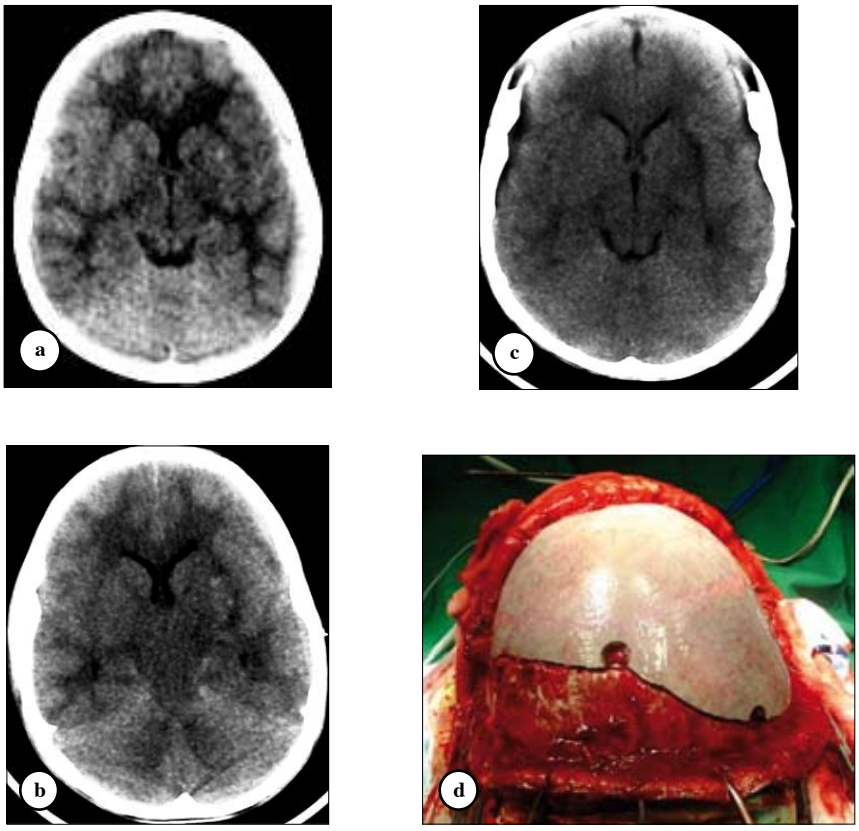

Figure 1 - **a)** Initial CT scan showing a small left frontal acute subdural hematoma and left caudate petechial brain contusion. **b)** CT scan after deterioration of the patient showing severe brain swelling, compression of the ventricles, and obliteration of basal cisterns. **c)** CT scan carried out 3 weeks after bifrontal decompressive craniotomy showing subsidence of brain edema, and absence of brain infarction. **d)** Intra-operative photograph taken during replacement of bone flap.

SHI and fracture left femur. The GCS on admission was 6/15, his pupils were unequal, and had sluggish reaction to light. After adequate resuscitation and stabilization of the patient, CT brain scan showed a small left frontotemporal acute subdural hematoma, evidence of diffuse axonal injury, and mild brain edema (**Figure 1a**). The patient was admitted to ICU and managed according to standardized protocol; 36 hours later, his GCS dropped to 4/15, and the pupils became dilated and fixed. A bolus of mannitol 20% was given and urgent CT brain scan showed severe brain edema, compression of the lateral ventricles, and obliteration of the basal cisterns; however, there was no change in the size of the SDH (**Figure 1b**). The patient was taken straight to theater for BDC and insertion of external ventricular drain (EVD). The postoperative CT scan and bone flap are shown in (**Figures 1c & 1d**). Immediately after surgery, the ICP was less than 10 mm Hg and maintained within the normal levels, and the pupils became small and equal but were non reactive to light for a few days. The patient's recovery was quite dramatic, and he was discharged from the hospital after 9 weeks; at the time of discharge he was conscious and oriented, GCS 15/15, had right side weakness, but he was able to walk unassisted. The follow up period was 4 years; he attended school 12 months later and his performance was good, and the right side weakness had recovered with mild residual hand apraxia.

Patient 2. A 54-year-old lady who is known to have hypertension and hypothyroidism, she presented with aneurysmal SAH (Hunt and Hess grade II). Four vessel cerebral angiography revealed 2 ophthalmic aneurysms on the left internal carotid artery (**Figure 2a**). Craniotomy and clipping of the aneurysms was carried out on the third day after SAH. On the 2nd postoperative day, she developed severe cerebral vasospasm and her GCS dropped to

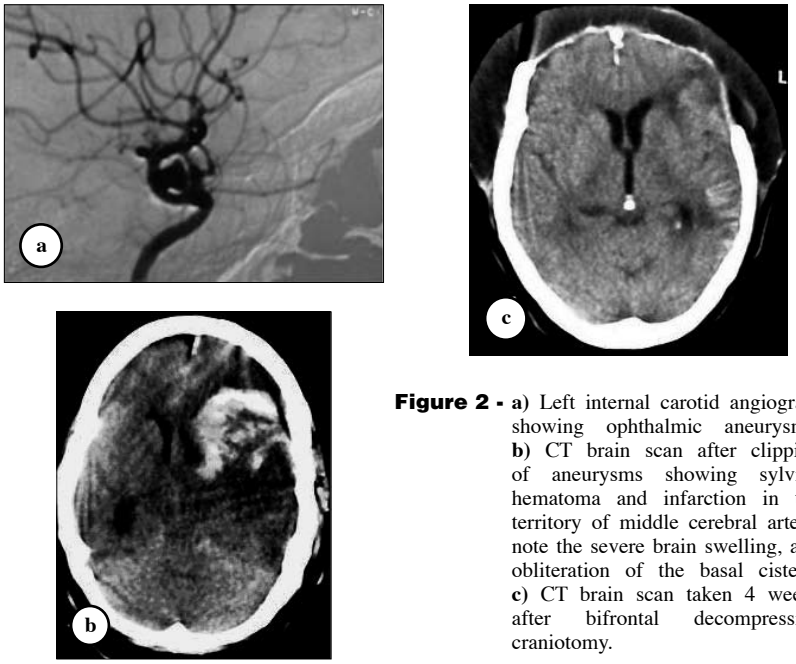

Figure 2 - **a)** Left internal carotid angiogram showing ophthalmic aneurysms. **b)** CT brain scan after clipping of aneurysms showing sylvian hematoma and infarction in the territory of middle cerebral artery, note the severe brain swelling, and obliteration of the basal cistern. **c)** CT brain scan taken 4 weeks after bifrontal decompressive craniotomy.

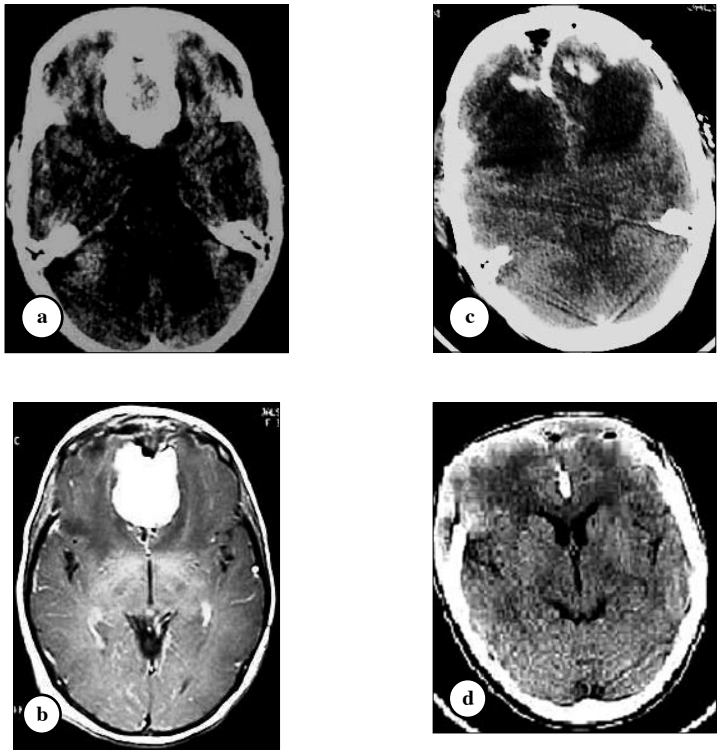

Figure 3 - **a)** Non contrast CT brain scan showing large heavily calcified olfactory groove meningioma. **b)** Post gadolinium T1 MRI scan showing the tumor, note the severe brain edema. **c)** CT brain scan 20 hours after tumor excision showing severe brain swelling, and complete obliteration of the basal cisterns. **d)** CT brain scan 2 weeks after bifrontal decompressive craniotomy.

9/15. Emergency CT brain scan showed hematoma in the sylvian fissure, marked swelling of the left hemisphere, and hypodensity in the territory of the middle cerebral artery suggestive of brain infarction. Despite aggressive treatment, the patient's condition deteriorated further to GCS 5/15 and her pupils became dilated and fixed. Repeat CT scan showed a well-established left hemisphere infarction, severe brain edema, compression of the lateral ventricles, obliteration of basal cisterns, and loss of gyral pattern (**Figure 2b**). Bifrontal decompressive craniotomy was performed as last resort therapy. The postoperative period was stormy and was complicated with extensive lower limb deep vein thrombosis (DVT), and chest infection requiring prolonged ventilation and tracheostomy. Despite the complicated postoperative course and significant brain damage, the patient's recovery was dramatic; she was weaned off ventilation in 3 weeks time and transferred to the neurosurgery ward one week later, follow up CT scan carried out after 4 weeks is shown in **Figure 2c**. The total period of hospital stay was 60 days; at the time of discharge she was conscious but dysphasic, she had right side hemiparesis but able to walk with assistance, and the bone flap was replaced before her discharge from hospital. The patient had regular follow up for 4 years; she had significant cognitive impairment in the form of attention and memory deficit, however, she was able to communicate with people, the hemiparesis had completely recovered, and the patient could take care of herself at home with occasional assistance (Karnofsky score 60).

Patient 3. A 22-week pregnant lady, G3, P2+0, presented with recurrent attacks of grand mal seizures and deterioration of her consciousness. On admission, her GCS was 13/15, she had bilateral papilledema but no focal neurologic deficits, obstetric assessment and abdominal ultrasound confirmed a single viable fetus. Radiological investigations, CT and MRI scans showed a large heavily calcified olfactory groove meningioma with massive peritumoral brain edema (**Figures 3a & 3b**). The patient's condition had dramatically improved on dexamethasone, and her seizures were adequately controlled with phenytoin. The patient was discharged on a tapering dose of steroids and scheduled for elective cesarean section at the end of the 36th week of pregnancy. One week prior to the planned day of delivery, the patient developed status epilepticus and she had an emergency cesarian section; this was uneventful and the baby was normal. Six weeks after delivery she had craniotomy and total excision of tumor. Postoperatively, the patient was electively kept on mechanical ventilation, because of the marked brain edema seen on the preoperative MRI scans. Twenty hours after craniotomy the patient's condition suddenly deteriorated to posturing, and her pupils became dilated and fixed. Urgent CT brain scan showed severe brain edema, compression of the ventricles, obliteration of the basal cisterns, and loss of gyral pattern (**Figure 3c**). The patient was taken immediately to theatre for BDC and duraplasty; immediately after surgery her pupils became small and equal, and by the next morning she was obeying commands and had no motor deficits, postoperative CT brain scan is shown in **Figure 3d**. The patient was kept on mechanical ventilation for 72 hours; on the 6th post operative day she was fully conscious, oriented (GCS 15/15) had no neurologic deficits and the bone flap was repositioned after 2 months. The patient was followed up regularly for 4 years, she had no neurologic deficits, no tumor recurrence or complications related to the bone flap, and she restarted her previous job as teacher after 4 months.

Discussion. Bifrontal decompressive craniotomy with duraplasty has been performed as last resort treatment for patients with intractable cerebral edema caused by trauma. Other types of decompressive craniectomy such as unilateral or bilateral frontal and subtemporal decompression, or circumferential hemicraniectomy has been described for patients with SAH, brain infarction, and subdural empyema.[11-13] From the pathophysiologic point of view, BDC provides more rapid and effective control of raised ICP than other types of decompressive surgeries for of the following reasons; it lowers the ICP to its normal levels immediately, it adds a vector of expansion to the cerebral hemispheres, which relieves subfalcine and transtentorial brain herniation, and allows exploration of the subdural space on both sides. In addition, it allows quick tapering of the medical treatment such as; hypothermia, barbiturates, osmotic diuretics, ventriculostomy, prolonged hyperventilation, and hypertonic saline, in order to avoid its potential complications.[14-17] In the present series, BDC was used for patients with malignant brain edema caused by different etiologies, trauma, aneurysmal SAH, and following excision of meningioma. All patients except one survived, their pupils became small, and the ICP was reduced to its normal levels immediately following surgery. The literature is deficient in reports of BDC for treatment of intractable cerebral edema following excision of brain tumors. The present study included one patient (patient 3) who developed intractable brain edema and herniation syndrome following excision of a large and heavily calcified olfactory groove meningioma. The patient had made an excellent and rapid recovery after

BDC. The previous experience with similar situations was bad; patients either died or developed massive brain infarction and ended up with severe disabilities despite aggressive medical treatment, removal of craniotomy flap, and brain excision, which was used by other authors[18] to resuscitate such patients. Smith and colleagues,[19] performed wide decompressive frontotemporoparietal craniectomy and duraplasty together with aneurysm clipping for 8 patients who had large sylvian hematoma caused by ruptured middle cerebral artery aneurysm and had poor Hunt and Hess grade (VI and V). They reported immediate decrease in ICP postoperatively, and the outcome was good in 5 patients, fair in one, poor in one, and one patient died. In the present series, BDC was performed in 2 patients with aneurysmal SAH who developed severe vasospasm and brain infarction, which was massive and involved the dominant hemisphere in one (patient 2). The recovery of both patients was protracted; both of them had initially severe disability but were not vegetative. One of them had improved to independence in 2 years time. The poor outcome of these patients highlighted the importance of performing craniotomy before development of brain infarction

The outcome of BDC has varied from one report to another; the percentage of good outcome has ranged from 7-70%, and mortality from 13.5-90%.[8,11,13,15,17,19] This wide variation in outcome of BDC could be attributed to the amount of primary brain damage, neurologic status (GCS) after resuscitation, and the timing of surgery. The poor outcome in early reports of BDC has withdrawn the interest in the procedure for sometime, however, it is getting more popular and reports of favorable outcome following surgery are increasing in the last decade. Whitfield and colleagues[20] in 2001, reported 69% as good outcome after BDC, 8% severe disability, and 23% mortality in a series of 26 patients with posttraumatic refractory IH, they provided pathophysiologic evidence that BDC had significantly reduced IH and improved pressure dynamics in head injury patients.

The timing of BDC is considered as an important predictor of outcome; ideally, the procedure should be carried out before the evolution of brain infarction and development of secondary brain damage. Polin and colleagues,[21] reported 57% rate of favorable outcome in a group of patients (12 of 35 with posttraumatic refractory IH) who had BDC carried out within the first 48 hours of injury, and before ICP values have exceeded 40 mm Hg for a sustained period. The overall rate of favorable outcome was 37%. They have shown better results of BDC over medical treatment in the control group obtained from Traumatic Coma Data Bank. Marshall[22] had also recommended BDC, if the ICP is more than 25 mm Hg for periods more than 30 minutes. Several factors including clinical status in the field and emergency room, GCS, pupillary responses, elevation of ICP, severity of brain injury, and the presence and severity of extracranial injuries have been demonstrated to be strongly associated with outcome after head injuries. Of the previous factors, the amount of primary brain injury, timing of surgery, level of ICP, and GCS score before surgery are considered as predictive of outcome after BDC.[8,11,13,15,17,19-22]

The present series, including 10 patients who underwent BDC for intractable cerebral edema caused by different etiologies, has demonstrated a clear benefit of surgery with 60% good outcome (GOS 5), and 90% survival rate. This has stimulated the proposal of the following criteria for good outcome after BDC; initial GCS >5, small pupils on admission, observed clinical deterioration, rapid surgical interference and absence of brain infarction in the preoperative scans. Although very difficult to accomplish, a randomized clinical trial is necessary to define criteria for surgical interference in patients with intractable cerebral edema.

In conclusion, BDC provides an effective and rapid control of ICP in patients with intractable brain edema and refractory IH regardless of the etiology. The procedure should be performed quickly following clinical deterioration, and before the development of irreversible damage.

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