Decompressive hemicraniectomy in malignant middle cerebral artery infarction: outcomes of 22 patients

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Abstract

Large middle cerebral artery (MCA) infarctions presenting as malignant cerebral edema account for 1-10% of all ischemic strokes. Mortality rates have been reported to be as high as 80%, and most of the survivors are left severely disabled. Decompressive hemicraniectomy (DHC) can reduce mortality and severe disability. The objective of this work is to help better define the selection criteria for performing the surgery in case of large MCA infarctions.

From August 2000 to March 2014, 22 patients with malignant MCA infarction were included in the study. During that period, all the patients underwent DHC due to transtentorial herniation despite the appropriate medical treatment. The mean age of the patients was 63 years old. Among them, 9 patients (41%) were expired.

The outcomes of patients with malignant MCA infarction who underwent DHC were compared with the previously published data. In the management of patients with acute ischemic stroke, it has become clear that in a number of these cases a progressive and often fatal deterioration secondary to mass effect from the edematous, infarcted tissue occurs. Some of these patients may benefit from undergoing a DHC but the timing and indications for this potential lifesaving procedure are still debated. DHC may be an useful procedure in patients with malignant MCA infarction. Timing of surgery and appropriate patient selection based on age and other criteria requires further investigation.

KEY WORDS: brain edema, cerebral infarction, decompressive craniectomy, middle cerebral artery.

Introduction

The middle cerebral artery (MCA) is the biggest of the intracerebral vessels and supplies through its pial branches almost the entire convex surface of the brain, including the lateral frontal, parietal, and temporal lobes; insula; claustrum. The lenticulostriate branches of the MCA supply the basal ganglia, the lateral parts of the internal and external capsules, and sometimes the extreme capsule. Occlusion of the MCA usually occurs in either the main stem (M1) or in one of the terminal superior and inferior divisions (M2). Occlusion of the M1 segment of the MCA prior to the origin of the lenticulostriate arteries in the presence of a good collateral circulation can give rise to the massive striatocapsular infarct. Occlusion of the MCA is the most common type of anterior circulation infarct, accounting for approximately 90% of infarcts and two thirds of all first strokes. Of MCA territory infarcts, 33% affect the deep MCA territory, 10% affect superficial and deep MCA territories, and over 50% affect the superficial MCA territory (1). Some patients with acute, nearly complete MCA or panhemispheric infarction may develop massive concomitant oedema with significant midline shift or compression of the basal cisternae, resulting in clinical signs of uncal herniation, a condition named malignant MCA infarction (2).

Life-threatening, complete middle cerebral artery (MCA) infarction account for 1-10% of all ischemic strokes. In this subset of patients, the rates of mortality range from 80 to 90% (3, 4).

Brain edema was revealed within the first day after onset of symptoms involving the grey and white matter surrounding the infarcted tissue. Swelling was at its greatest on days 3 to 5, with oedema decreasing within 2 weeks. Severe brain swelling after cerebral infarction is known to originate transtentorial and uncal herniation, with clinical deterioration and progressive brainstem dysfunction. The patients rarely survive and, if they do, severe disability or a vegetative state is frequent (2).

Cerebral oedema due to ischemia is initially cytotoxic, delineate by intracellular water accumulation, and later vasogenic, in which water moves across the blood-brain barrier (BBB) into the extracellular interstitial space. The interruption of the BBB can be demonstrated as early as 20 minutes after transient global forebrain ischemia in rats and is critically determined by small variations in intraischemic brain temperature (5). Cerebral ischemia results in oedema development in and around the ischemic area, the larger the area of the infarction, the greater the extent of oedema. In the case of malignant MCA infarction, the entire vascular distribution of the MCA, and possibly the anterior...
cerebral artery, is compromised. Oedema is responsible for the parenchymal hypodensity that is exhibited on computed tomography (CT) scanning (Figure 1). One of the principal pathophysiological processes after cerebrovascular stroke is the development and propagation of an escalating cycle of brain swelling and an increase in intracranial pressure (ICP). The goals of the clinical management consist of suspending this cycle by controlling ICP and maintaining cerebral perfusion pressure and cerebral blood flow to avoid brain ischemia. Progressive brain oedema and the exacerbating effect it has on increasing ICP can cause the area of damaged brain to enlarge. Within the delineated cranial vault, the oedematous tissue places pressure against surrounding normal parenchyma. Intracranial hypertension results in decreased cerebral perfusion pressure and therefore decreasing blood supply throughout the brain. Because of the increase in ICP, other major cerebral vessels may be compressed by the expanding tissue, against dural edges or against the skull. The result is secondary ischemia and a further extension of the infarcted area (6). Clinically, the patients have a serious hemispheric syndrome with head turning and eye deviation. Early somnolence and respiratory disorder occur. Somnolence may be present as early as 3 hours after stroke onset. If the dominant hemisphere is involved, global aphasia is always present. Generally, these patients show a rapid decline in consciousness and develop the signs of herniation 2 to 4 days after onset of symptoms. Most patients need intubation and artificial ventilation. During the farther clinical course, failure of medical treatment for elevated ICP occurs. Once the ICP has moved into critical values (>20 mm Hg), both clinical appearance and CT already show signs of cerebral herniation.

Brain death generally occurs between days two and five after onset of stroke (2). Early death is a result of transtentorial herniation while delayed death is typically due to medical complications of prolonged hospitalization containing pneumonia and pulmonary emboli (7). The factors that define degree and speed of ischemic brain oedema are not well known. Proximal vessel occlusion, poor collaterals, hypothermia or hyperthermia, electrolyte disturbances, late reperfusion, and extent and distribution of the ischemic area, may influence malignant brain swelling. The time course of brain swelling is not well known too.

Most patients develop signs of herniation between day 2 and day 4 after onset of stroke (2). Optimal medical management for malignant oedema due to MCA infarction has not been standardized. In the HAMLET (Hemicraniectomy After Middle cerebral artery infarction with Life-threatening Edema Trial), recommendations for medical management of stroke-related malignant oedema included osmotherapy, admission to the intensive care unit, invasive monitoring of ICP, blood pressure control, elevation of the head to 30°, and maintenance of normothermia, normoglycemia, and normovolemia (8).

Systemic hypothermia (target temperature 33-35°C) for ICP control can be obtained with ice packs, intravenous cold saline solutions, cooling blankets, surface and intravascular cooling devices. There are a few safety trials, but no efficacy studies, despite reliable demonstrations of reduced infarct volume with hypothermia after ischemic stroke in animal models (9). Proposed as a life-saving procedure, experimental and clinical evidence indicates that an early decompressive hemicraniectomy (DHC) can limit the extension of the infarcted area. From a mechanical perspective, DHC provides an immediate opening in the otherwise closed cranial vault. There is an immediate decrease of ICP. Therefore, compression of normal tissue is prevented or circumscribe. The additional space created allows the tissue to expand through the bone defect, away from midline structures; herniation is minimized or completely solved postoperatively (Figure 2).

A decrease in ICP support an increase in cerebral perfusion pressure, aiding blood flow to the ischemic area, optimizing circulation to the damaged area through collateral vessels. Because DHC alone may improve blood flow in the ischemic area, surgical resection of the infarcted tissue should not be conducted in these patients (6). An adequate DHC needs to cover a large surface area, ideally beyond the margins of the ischemic area in all directions. The bone flap includes frontal, temporal, and parietal bone. The limiting factor is its anterior-posterior diameter, as
the vertical diameter cannot exceed 9-10 cm from the floor of the middle cranial fossa to the superior sagittal sinus.

The target diameter should be 14 cm anterior-posterior, temporal bone needs to be resected down to the middle cranial fossa. An increase in the anterior-posterior diameter from 12 to 14 cm, product an increase of potential volume by 76%.

A small diameter DHC leads to compression and kinking of bridging veins, or mushroom-like herniation of the brain parenchyma with shearing distortion and supplementary ischemic lesions.

After removal of the temporal bone to the skull base, the dura is opened, adjusted, and a dural patch is placed into the incision (duroplasty) to prevent leakage of cerebrospinal fluid (CSF).

The bone flap can be conserved in the abdominal subcutaneous tissue or frozen.

Reimplantation of the bone flap (cranioplasty) is possible 6-12 weeks up to 6 months after DHC, once the swelling has resolved.

DHC was found to be well tolerated after intra-arterial thrombolysis.

Periprocedural complications consist of intracranial, wound and bone flap infection, subdural and epidural hematoma, contralateral subdural effusions, and hypotension with the risk of supplementary ischemic lesions.

Late complications consist of extra-axial fluid collections, hydrocephalus, chronic pain, subdural hematomas and sinking skin flap syndrome (Figure 3).

The principal symptoms of sinking skin flap syndrome (or “syndrome of the trephined”) are severe orthostatic headache, mental changes, focal deficits, or seizures. The presumed cause of the sinking skin flap syndrome (SSFS) is the pressure gradient between the atmosphere and the intracranial vault that may be aggravated by CSF diversion, CSF hypovolemia, dehydration, and position change.

Those patients tend to have a smaller surface of the craniectomy with larger lesion size which leads to increased areas of atrophy, and a longer delay to cranioplasty.

The SSFS may evolvement to “paradoxical herniation” as a consequence of the atmospheric pressure exceeding intracranial pressure and may eventually lead to neurological deterioration, coma or death.

Treatment includes placement in supine position and cranioplasty as soon as possible (9).

Although many physician are reluctant to offer DHC to patients with dominant hemisphere infarcts, a meta-analysis found no difference in functional outcome comparing left versus right-sided (10).
Medical complications (pneumonia, gastrointestinal bleeding, and sepsis) due to patient immobility and survival from the DHC can influence the clinical outcomes of the patient. Although DHC has led to a significant decline in mortality rates, several problems remained to be solved. Many of them resulted from the loss of bony covering and the changes associated with it. A decompressive surgery that used a technique allowing the bony flaps to be open gradually depending on the intracranial pressure would theoretically resolve many of the problems related to DHC.

The idea of performing a “window-like” craniotomy as an alternative to substitute DHC was reported in 2003 by Valença (11).

The surgical technique of the “window-like” craniotomy involves a large semi-circular skin incision starting in the proximities of the midline and extending to the posterior parietal area, ending at the level of the tragus. The scalp must be elevated from the underlying pericranium to provide greater elasticity and a looser skin flap to allow the stitching up of the skin at the end of the “window-like” procedure, covering again the bone with the skin.

An extensive, approximal rectangular-shaped craniotomy is performed involving frontal, temporal, and parietal bones, and part of the occipital squama (diameter 12-15 cm). The angle of the bone cut must be bevelled outwards to allow the upper part of the craniotomy bone flap to rest on the adjacent skull and prevent penetration into the intracranial cavity. An anterior temporal craniectomy (sub temporal decompression) may be added to relieve temporal lobe pressure. Dural incisions are made and the dura is fixed at the bone border to prevent epidural bleeding (Figure 4).

A dura patch is placed in the incision. Using a vertical cut, the bone flap is divided into two equivalently sized pieces, which will be the opening of the “window lids” (Figure 5).

The outer frontal and parietooccipital sides of the flap are each one tied to the skull at two points using a synthetic nonabsorbable suture to function as a hinge joint that allows opening of the window but avoids downward movement of the bone inside the skull (Figures 6, 7).

After a few months of follow-up the appearance of the “window-like” craniotomy was similar to that of a normal classical cranioplasty, with no signs of movement of the partially free bone flaps (Figures 8, 9).

The proposal of decompressive surgery using a craniotomy in a “window-like” fashion presents the following advantages:

1. it allows the oedematous cerebral parenchyma to herniate with a gradual opening, simultaneously relieving the elevated ICP;
2. it may avoid the development of SSFS;
3. it obviates the need for a cranioplasty at a later stage, which may have an impact on the overall treatment cost;
4. it is not necessary the storage of a large bony flap under the abdominal fat, a procedure that is time-consuming and is cause of moderate to severe abdominal pain (pain causes agitation thereby increasing intra-abdominal and intracranial pressures), sometime associated with massive hematomas and infection.

The “window-like” craniotomy allows the performance of a large hemicraniectomy to adequately decompress the ischemic brain and avoid hemispheric lesions. At the same time, it offers an anatomic, economical solution that allows the gradual accommodation of the herniated brain tissue, with a decrease in the ICP after brain insult.

The principle of the window-like craniotomy mimics the opening of the sutures observed in child hydrocephalus. On the other hand, by recomposing a resistant barrier between the brain parenchyma and the
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Figure 6 - Left side “window-like” craniotomy. The outer frontal and parieto-occipital sides of the flap are each one tied to the skull at two points using a synthetic nonabsorbable suture to function as a hinge joint that allows opening of the window but avoids downward movement of the bone inside the skull.

Figure 7 - Left side “window-like” craniotomy that will progressively open when a transcalvarial herniation occurs, to decrease the ICP and relieve brainstem compression.

Figures 8, 9 - 3D head CT scans, subsequently normalization of the ICP, showing good alignment of the craniotomy flaps on the adjacent skull after brain decompression with the “window-like” craniotomy.
environment, it possibly prevents the SSFS (12-14). With increasing availability of DE as an aggressive treatment option, identification of predictors of a malignant course of the MCA or ICA infarction is extremely important. The involvement of more than 50% of the MCA territory and a perfusion deficit of more than 66% on CT, are the most reliable predictors of oedema generation (15). The aims of this study were to determine survival, prognosis and functional outcome within a series of consecutive 22 patients who underwent DHC for treatment of malignant MCA infarction.

Materials and Methods

From August 2000 to March 2014, 22 patients (11 men, 11 women) with a mean age of 63.27 years (range, 45-76 years) were included in this study. The demographic data of patients are presented in Table 1.

All patients underwent CT scan immediately prior to surgery. 21 of them underwent DHC, only the last one of the series (D.C.) was underwent "window-like" craniotomy. Inclusion criteria were:

1. infarction of >50% of the MCA territory as measured by CT scan, with an acute onset of corresponding clinical signs and symptoms like pupillary dilatation, decline in the level of consciousness, respiratory failure and aggravated neurological signs;
2. neuroradiological evidence of local brain swelling such as midline shift of 5 mm or more indicating space occupying oedema.

Clinical status was rated on admission and at surgical decision time using the National Institute of Health Stroke Scale (NIHSS) (16) that was repeated one week after surgery. Patients were revalued 12 months after surgery and the outcome was quantified on the modified Rankin scale (mRS) (17). The twelve-month

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functional outcome was dichotomized between mRS score 1 to 4 (favourable outcome) versus 5 to 6 (poor outcome).

Results

Eight patients (36%) undergoing surgery had a cerebral infarction in the nondominant hemisphere. Twenty-one patients (95.46%) did not fulfill the criteria for thrombolysis according to standard therapy guidelines. Thrombolysis was attempted intra-arterially in one (4.54%) patient (D.C.): this patient did not improve clinically.

Stroke aetiology were large-vessel atherosclerosis in 9 patients (41%), cardio embolic in 7 patients (31.8%). Seven patients had a history of atrial fibrillation (31.8%); 7 (31.8%) had diabetes mellitus; 15 (68%) had hypertension.

The mean time between onset of symptoms and surgery was 95 hours (range 23 to 309). Five of 22 patients died despite aggressive medical management and DHC within 7 days from surgery. The cause of death in all cases was intractable ICP due to infarction progression.

Four patients died later, within 71 days from surgery. The one-month mortality rate was 31.8%, and the six-month mortality rate was 41%.

The overall incidence of surgical complications with the procedure was 22.7%.

In one patient postoperative subdural hygroma was diagnosed and evacuated.

In one patient postoperative SSFS was diagnosed and treated with cranioplasty.

Three patients (13.6%) underwent ventriculoperitoneal shunting after communicating hydrocephalus was confirmed as a cause of neurological deterioration. The mean initial NIHSS score for all patients was 20.4.

In the group, 6 patients (27.27%) were older than 70 years of age, and 16 (72.73%) were younger than 70. There were some imbalances in the patients’ characteristics.

The mean initial NIHSS score in the group older than 70 years of age was 25.8. The mean initial NIHSS score for patients younger than 70 years was 17.3; favourable functional outcome at 12 months after surgery was noted in 9 patients (56%) in this group.

In the group older than 70 years of age, unfavourable outcome was noted in all patient; the one-month mortality was 33%, while unfavourable functional outcome at 12 months after surgery was noted in 4 patients (67%).

Discussion

In 2006 and 2007, data from three randomised trials were published providing substantial evidence for a dramatic reduction in mortality with DHC. The trials were: DEcompressive Surgery for the Treatment of malignant INfarction of the middle cerebral artery (DESTINY) (18), DEcompressive Craniectomy in MALignant middle cerebral artery infarcts (DECIMAL) (19), and Hemicraniectomy After Mid- cerebral artery infarction with Life-threatening Edema Trial (HAMLET) (20).

DESTINY was an open, controlled, prospective, multicentre, randomised trial. Patients were randomised to either surgical plus conservative treatment or to conservative treatment alone. The maximum time from symptom onset to treatment start was 36 hours. All patients were treated in an intensive care unit (ICU) and were intubated and ventilated. It was based on a sequential design, taking mortality after 30 days as the first end-point, and randomisation was planned to go on until statistical significance for this end-point was reached. Consequently, patient enrolment would be interrupted until the six-month functional outcome end-point (primary end-point) – mRS dichotomised at a score of 0-3 versus 4-6 – had been collected. Depending on the observed difference in functional outcome, the final sample size would be recalculated for a second explorative trial stage. Secondary end-points included analysis of the mRS 0-4 versus 5-6 and the distribution of scores of the mRS at six months and at one year. After inclusion of 32 patients between February 2004 and October 2005, patient recruitment was stopped due to the statistically significant results of mortality: in the intention-to-treat analysis, 2 of 17 patients (11.8%) treated by DHC had died, whereas 7 of 15 patients (50.3%) who received maximum conservative treatment on the ICU alone had died after 30 days (p=0.02). Functional outcome data after 12 months showed that 47.1% of the patients in the surgical arm and 26.7% of the patients in the conservative arm reached an mRS of 0-3 (p=0.23), and 76.5% in the surgical arm versus 33.3% in the conservative arm reached an mRS of 0-4 (p=0.01). Analysis of the distribution of the mRS scores showed positive results in favour of surgery (p=0.04). After a sample size projection for the primary end-point suggested a number of 94 patients to be included in each arm, the trial was stopped.

DECIMAL was an open, controlled, prospective, multicentre trial that also randomly assigned patients to either surgical plus conservative treatment or to conservative treatment alone. Among other criteria, an infarct volume on diffusion-weighted imaging (DWI) of at least 145 cubic centimetre (cc) qualified patients for inclusion. DHC had to be performed within 30 hours after symptom onset and within 6 hours after randomisation. The primary end-point in DECIMAL was functional outcome based on the score on the mRS, dichotomised 0-3 versus 4-6. A sequential design for this end-point was chosen based on interim analyses after every 4 patients. Secondary end-points included survival and the score on the mRS at 6 and 12 months. Between December 2000 and November 2005, 38 patients were enrolled. Survival was significantly dif-
Recovery in young patients surviving a severe stroke

Recovery after ischemic stroke generally follows a nonlinear pattern, with the highest rate of recovery in the first weeks, and limited improvement after 6 months. HAMLET found improvement in activities of daily living and quality of life after DHC between 1 and 3 years after stroke onset. However, this large reduction in case fatality comes at the expense of an increased risk of moderately severe or severe disability at 1 year, and the majority of survivors have global cognitive impairment; some Authors have therefore expressed concerns about the effect of decompressive surgery on long-term quality of life (21, 22). In HAMLET adult patients ≤60 years of age with space-occupying hemispheric infarction were randomly assigned to DHC or to best medical treatment. The primary outcome measure was functional outcome as measured with the mRS at 1 year, dichotomised between good (mRS, 0-3) and poor (mRS, 4, 5, or death). Predefined secondary outcome measures included functional outcome at 3 years, and case fatality, functional dependence assessed with the Barthel Index, quality of life estimated with the Medical Outcomes Study 36-item short-form health survey and a visual analogue scale, symptoms of depression measured by the Montgomery and Åsberg Depression Rating Scale, and caregiver strain assessed with the caregiver strain index at 1 and at 3 years.

HAMLET shows that the effects of DHC on case fatality and functional outcome in patients with space-occupying hemispheric infarction are sustained for up to 3 years. DHC reduced the risk of death at 1 and 3 years but had no effect on the chance of a good functional outcome. Quality of life improved between 1 and 3 years in patients treated with surgery. Previous reports on outcome in randomized trials of DHC for space-occupying hemispheric infarction were limited to the first year after stroke.

Meta-analyses that have demonstrated a benefit of DHC were limited to treatment in the first 48 hours. In HAMLET, patients could be enrolled within 96 hours after stroke onset. Recovery after ischemic stroke generally follows a nonlinear pattern, with the highest rate of recovery in the first weeks, and limited improvement after 6 months.

HAMLET found improvement in activities of daily living and quality of life after DHC between 1 and 3 years after the stroke. Recovery in young patients surviving a severe stroke apparently may take a long time, and outcome assessments in these patients should probably not be limited to the first year (21).

It has still not been clarified whether to operate as soon as possible, when the diagnosis of malignant MCA infarction has been made, or to wait for development of clinical deterioration, midline shift on brain imaging, increased intracranial pressure or signs of herniation. In fact malignant MCA infarction does not invariably result in fatal brain oedema. There are patients with large brain infarctions who rapidly develop fatal brain swelling. It is important to recognize those patients who are at risk of developing a malignant clinical course as soon as possible. In these patients early DHC is presumably the only life-saving procedure.

On the other hand, there are patients with massive ischemic stroke but only mild brain swelling over a long period of time. Many of these patients never develop signs of herniation, and DHC may not be required. It is uncertain which factors promote early and rapid brain swelling and which factors are protective. Experimental studies have suggested that reperfusion of already irreversibly damaged brain parenchyma may enhance oedema formation.

Controversy remains with regard to the question of whether patients with malignant MCA infarction of the dominant hemisphere should receive DHC. The loss of ability to communicate in association with severe hemiplegia was considered to be too disabling, and DHC was often restricted to patients with a non-dominant hemispheric infarction. From the randomised trials and larger prospective case series there is currently no indication that patients with dominant malignant infarctions do not benefit from DHC. Neither mortality nor functional outcome was associated with the hemisphere; indeed, the impairment caused by aphasia may be balanced by the neuropsychological deficits from which patients with infarction of the non-dominant hemisphere suffer, i.e. severe attention deficits, apraxia and depression. In addition, the long-term aphasia in dominant malignant MCA infarction is rarely global.

Till now there is no evidence that DHC should not be considered for patients with dominant hemisphere MCA infarction (23). Usually there is an unfavourable outcome in older patients after DHC, thus many physicians recommend an age limit between 50 and 60 years. In the analysis from Gupta et al., age was the only prognostic factor for a poor outcome (10). At present, the available data cannot define an age limit after which DHC should not be performed. Our data suggest an age limit of 70 years, but further studies are necessary.

Although DHC is firmly established in the management of malignant MCA infarction (24), there remain some clinical and ethical questions that need to be addressed in the future.

Optimal timing of DHC for MCA infarction is un-
known; analyses of data from HAMLET suggest that delaying >48 hours does not realize outcome benefit, but further studies are necessary. As progress in endovascular techniques and early reperfusion occurs, the association of endovascular and surgical treatments of strokes will need to be studied.

Alternative procedures to the classic DHC, including “window-like” craniotomy. Theoretically, the “window-like” craniotomy allows the performance of a large hemicraniectomy to adequately decompress the brain and avoid hemicraniectomy-associated lesions. Moreover, the “window-like” craniotomy adds the advantage of avoiding a second surgical procedure (cranioplasty), but further studies are necessary to confirm the data.

In the end, novel therapeutics for the management of stroke-related oedema could replace or complement DHC in ischemic stroke.

References