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Decompressive Craniectomy for Refractory Intracranial Hypertension

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1. Introduction

Clinical and experimental data from the past two decades show that Decompressive Craniectomy (DC) is an effective treatment which reduces mortality within patients with refractory intracranial hypertension. Massive cerebral ischemic infarction and traumatic brain injury are the most frequent indication of DC. Since the conservative medical treatment of intracranial hypertension is ineffective in many patients, the idea of decompressive surgery of temporary release of swollen brain outside the cranium has been developed at the beginning of the last century. The first decompressive hemicraniectomy for traumatic brain injury was done in 1901 by Kocher. (Merenda & DeGeorgia, 2010) Harvey Cushing started using DC for the treatment in the cases on inoperable brain tumors and later also in the cases of traumatic diffuse brain edema and vascular malformations. (Kahar et al,2009) Decompressive surgery was first reported as a potential treatment for large hemispheric infarction in case reports as early as 1956. (Scarcella, 1956)

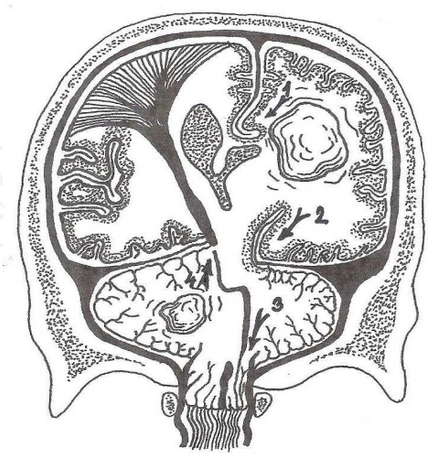
The results of experimental studies using rat models and prospective studies with acute stroke patients have provided further support for decompressive surgery strategy in patients with intracranial refractory hypertension. On the basis of these facts three randomized prospective studies with patients with malignant supratentorial infarction were started in the first decade of 21st century. The pooled analysis of these studies proved reduction of mortality without an increasing number of disabled people. Based on the pooled analysis of DECIMAL, HAMLET and DESTINY, the European Stroke Association (ESO) issued some new guidelines for malignant supratentorial brain ischemia treatment. (European Stroke Organization guidelines, 2008) The recommendations for the decompressive surgery for traumatic brain injury are not so unambiguous. (Servadei, 2011) DC is recommended in children patients in some specific situations and nowadays it is not recommended in adults routinely. Also in some other cases which lead to intracranial hypertension development, DC is performed only on the basis of the individual approach of the doctor to the patient, often after consulting the family. Generally, we can say that DC remains the only one option of intracranial hypertension (ICH) of various etiology treatment when the conservative treatment fails.

2. Patophysiology of intracranial hypertension

The syndrome of intracranial hypertension appears when the intracranial pressure (ICP) arises up to more than 20-25mmHg. Sustained ICP values of greater than 40-45 mm Hg indicate severe life-threatening state .The possible causes of increased ICP are shown in table No1. The high ICP reduces the cerebral blood perfusion and space occupying lesion causes mass effect which then leads to brain tissue displacements and herniation. There are four most common types of herniations; the subfalcial, temporal lobe tentorial (uncal herniation) , cerebellar – foramen magnum and cerebellar –tentorial herniation . (fig 1) Temporal uncal herniation and both types of cerebellar herniations can lead to compression of brainstem and a rapid alteration of consciousness , anisocoria , decerebrate posturing and alteration of breathing (atactic or cluster type of breathing) followed by apnoe and cardiac arrest in the end.

mass effect such as malignant ischemic stroke with edema, contusions, subdural or epidural hematoma, brain tumor etc.
generalized brain swelling without mass effect can occur in ischemic-anoxia states, traumatic brain edema ,acute liver or renal failure, hypertensive encephalopathy, status epilepticus etc.
increasing venous pressure can be due to venous sinus thrombosis or heart failure.
obstruction of cerebrospinal fluid flow or malfunction of its absorption can occur in hydrocephalus or in meningeal disease (e.g., infecious, carcinomatous or subarachnoidal hemorrhage).
idiopathic or unknown cause (idiopathic intracranial hypertension , pseudotumor cerebri)

Table 1. The causes of increased intracranial pressure (ICP)



Legend: 1.subfalcinal herniation 2. temporal lobe tentorial (uncal) herniation 3. cerebellar – foramen magnum herniation 4. cerebellar –tentorial herniation

Fig. 1. The types of cerebral herniation, mass shifts associated with a parietal lobe and cerebellar tumor (Adams & Victor, 1997)

3. Conservative treatment of intracranial hypertension

Several types of the conservative treatment for reducing intracranial hypertension of various causes to prevent midline shift or herniation have been proposed in the past decades such as management of the airway, breathing and circulation (ABCs), osmotherapy, sedation, steroids, hyperventilation , and induced therapeutic hypothermia . (Sankhyan, 2010; Jüttler et al 2007) Osmotherapy (glycerol and mannitol) has been tested in several randomized and nonrandomized clinical trials of acute stroke, but none of these proved its effect on the clinical outcome. A systematic Cochrane review of these trials in acute stroke suggests a favourable effect of glycerol treatment on short-term survival, but no long-term efficacy. (Hofmeijer et al, 2003) The lack of proven benefit on long-term survival does not support the routine use of glycerol and mannitol in patients with acute ischemic stroke. None of the randomized trials in patients with ischemic stroke which would prove efficacy on their favourable outcome has been carried out. So far none of these terapeutic conservative strategies are recommended on level A or B for the treatment of ICH in space occupying ischemic stroke.

In traumatic brain injury (TBI) the recommendations are summarized in the Brain Trauma Foundation Guidelines. (Bullock et al, 2006) Hyperosmolar agents currently in clinical use for TBI are mannitol and hypertonic saline. Mannitol is widely used and its use is advocated in two circumstances. First, a single administration can have short term beneficial effects, during which further diagnostic procedures (e.g., CT scan) and interventions (e.g., evacuation of intracranial mass lesions) can be accomplished. Second, mannitol has been used as a prolonged therapy for raised ICP. (Bullock et al , 2006). There is a level II evidence that mannitol is effective for control of raised intracranial pressure (ICP) at doses of 0.25 gm/kg to 1 g/kg body weight. Arterial hypotension (systolic blood pressure < 90 mm Hg) should be avoided. (Bullock et al, 2006)

Current evidence is not strong enough to make recommendations on the use, concentration and method of administration of hypertonic saline for the treatment of traumatic intracranial hypertension.

4. Indications

The most common diagnosis, where DC is performed, are ischemic stroke and traumatic brain injury. Less frequently DC has been successfully reported in relation with the treatment of refractory intracranial hypertension in other diagnosis such as intracranial venous thrombosis, subarachnoidal hemorrhage , spontaneus intracerebral hemorhage , encephalitis , tumours and in encephalopathy related to Reye´s syndrom. (Schimer et al, 2008) Generally there are no fixed threshold value for surgery such as intracranial pressure value, midline shift size, expansion volume size, perfusion pressure etc. The indication for surgery is in most cases based on the individual approach of the clinician towards the patient.

4.1 Decompressive cranietomy for acute stroke

4.1.1 Malignant supratentorial ischemic infarction

4.1.1.1 Rationale and randomized trials

The incidence of ischemic stroke in various European countries is between 183-349 /100 000 and e.g. in the Czech Republic it is 219/100 000. (Bamford et al 1990, Bar et al, 2010)

Generally, the massive hemispheric infarctions constitute approximately 5-10 % of all types of ischemic strokes and have a mortality rate of 50% to 80%. The prevalence of malignant supratentorial infarction with space-occupying edema is 1-10% of patients with territory of Middle Cerebral Artery (MCA) infarction. (Hacke et al, 1996) There is no clear evidence which patients with MCA ischemic stroke develop malignant infarction. Oppenhiem et al demonstrated that the infarction volume 145 cm3 and more on diffusion weighed images on magnetic resonance (DWI MRI) and the clinical status of more than 20 points in the National Institute of Health Stroke Scale (NIHSS) are strong predictors of malignant supratentorial infraction development. (Oppenhiem et al 2000)

The clinical picture of cerebral infarction in the territory of the middle cerebral artery is preliminary shown by a severe neurological deficit (severe hemiparesis or hemiplegia, gaze palsy, aphasia and or dysarthria). In the period of the next 2 to maximum of 5 days from the stoke onset, approximately 10% of patients develop brain edema. Mass effect subsequently leads to transtentorial uncal herniation and coning. In the clinical picture there occurs consciousness deterioration in the first place which is not typical for uncomplicated brain ischemia of MCA. In the another progression of the uncal herniation there develops unilateral (ipsilateral) hemiparesis (in the clinical picture there already dominates quadriparesis) with the ipsilateral and later also bilateral mydriasis. Another intracranial pressure increase leads to apnoe and cardiac arrest. Despite the best medical treatments such as hyperventilation, osmotherapy, barbiturate coma, and induced hypothermia, mortality is estimated to be between 50% and 78%. (Gupta et al, 2004)

There have been many studies published up until the year 2004 giving evidence of the benefit of decompressive hemicraniectomy in the reduction in mortality. Gupta et al analysed 15 studies with the total number of 129 patients who fulfilled the criteria for entering the analysis. In his analysis he proved the reduction in mortality of 25-30% in operated patients (Table 2). (Gupta et al, 2004)

Craniectomy reduced mortality in patients with malignant MCA stroke, but it was not still clear which patients may avoid severe disability after the procedure. These studies were not randomized and with retrospective design in most of them and therefore Cochrane´s review from 2002 concluded there was no evidence to recommend DC to treat intracranial hypertension following ischemic stroke. (Morley NC, 2002). Many other studies doubted the effect of decompression mainly in patients at their old age and with their left hemisphere affected. The predictors of the favourable outcome were not set and therefore it was not clear which patients should be candidates for decompressive surgery. These controversies and never ending discussion among stroke experts led to the start of three randomized studies in the end.

The clinical effect of decompressive surgery on functional outcome has been studied in three European studies; DECIMAL trial (Decompressive Craniectomy in Malignant Middle Cerebral Artery Infarcts), in DESTINY trial (Decompressive Surgery for the Treatment of Malignant Infarction of the Middle Cerebral Artery) and in HAMLET (Hemicranietomy after Middle Cerebral Infarction with Life-threatening Edema Trial). Besides above mentioned trials two other randomized studies were done in past decade (Table 3).

Author	Right MCA, n	Left MCA, n	Mean Age, y	Patients With Early Surgery, n (%)	Patients With Brainstem Signs, n (%)	Mean Time to Follow-Up, mo	Patients With Good Outcome, n (%)	Patients Died, n (%)
Carter et al	14	0	49	5(36)	14(100)	12	8(57)	3(21)
Walz et al	10	8	50	9(50)	NA	14	6(33)	6(33)
Leonhardt et al	26	0	50	11(42)	NA	12	11(42)	6(23)
Holtkamp et al	9	3	65	4(33)	0(0)	7	1(8)	4(33)
Delashaw et al	9	0	57	3(33)	7(78)	15	4(44)	1(11)
Rieke et al	26	6	49	8(25)	24(75)	13	16(50)	11(34)
Koh et al	4	3	45	NA	NA	7	2(29)	1(14)
Rengachary et al	3	0	31	0	3(100)	21	1(33)	0(0)
Kalia and Yonas	2	2	34	1(25)	2(50)	17	3(75)	0(0)
Young et al	1	0	59	0(0)	1(100)	9	0(0)	0(0)
Ivamoto et al	1	0	49	0	1(100)	7	1(100)	0(0)
Kondziolka et al	3	1	42	2(50)	4(100)	20	4(100)	0(0)
Gupta et al	5	4	53	2(22)	6(66)	8	1(11)	1(13)

Table 2. Summary of Case series of the Decompressive Craniectomy (Gupta analysis, 2004)

DECIMAL and DESTINY were stopped in 2006 because of the benefit of the surgery on mortality, but primary clinical end point (benefit for the patient with mRS less or equal to 3) failed. (Jüttler et DESTINY Study Group, 2007; Vahedi et Decimal investigators, 2007) HAMLET study was finished in 2009 with the conclusion that there is no evidence that this operation improves the functional outcome when it is delayed for up to 96 hours after the stroke onset. (Hofmeijer et Hamlet investigators, 2009)

In 2007 the results from the three European randomised controlled trials (DECIMAL, DESTINY, HAMLET) were pooled to obtain sufficient data to reliably estimate the effects of decompressive surgery not only on the reduction in mortality but also in order to increase the number of patients with a favourable outcome. As the favourable outcome was chosen mRS equal or less than 4 in spite the fact that in the most studies score mRankin <= 3 is accepted. Distribution of the modified Rankin score after 12 months between the group treated with and without decompressive surgery is shown in Table 4 .(Vahedi et al,2007) The favourable outcome defined in mRankin scale 0-4 has given rise to discussion again among neurologists and neurosurgeons. Many clinicians do not consider the state of the

patient rated in mRankin scale 4 as a favourable outcome. But decompressive hemicraniectomy based on the above studies was recommended in strictly selected patients in the European Stroke Organization guidelines 2008. (European Stroke Organization (ESO), 2009)

Authors	n	Design	Conclusions
Pooled analysis	93	Multicentre International RCT. 51 patiens	´Good outcome´ defined as MRS ≤4 10/42 (24%)
DECIMAL,		randomized to surgery along standard protocol/	MRS < 4 at 1 year in medical group, 22/51 (43%)
DESTINY and		technique,42 managed medically(Mean age	in surgical group
HAMLET		45.1)Main Inclusion criteria:age 18-60,>50%	Most patients randomized < 24 hours, Subgroup
(2007)		MCA infarct on CT,<45 hours symptom onset.	analysis not possible due to insufficient numbers.
		Exclusion criteria: Haemorrhage,MRS ≥ 2,	Largerst RCT to date
		expectancy < 3 years	
HeadDDfirst	26	Multicentre RCT.Inclusion: MCA infarct with	Stopped early due to very large difference in in 21 day
		clinical or radiological deterioration after 96 hours	mortality favouring surgical group. Publication
		of onset.Age < 75	pending
Mori et al.	71	retrospective analysis of massive hemispheric	6/12 Mortality in medical group 71.4%
(2004)		infarcts(volume > 200 cm3).Divided into 3	Late DC 27.6 % at 6/12
		groups-21/71 medical management alone.50/71	Early DC 19.1 % at 6/12
		underwent DC+Duroplasty subdivided into 21	6/12 outcome in suvivors revelead statistically
		´early´ DC and 29´late´ DC after clinical/	significant improved GOS in early DC vs
		radiological herniation	conservative. Little difference between late DC vs conservative.

Table 3. Randomized controlled trials on DC for malignant MCA infarction (Kakar et al, 2009)

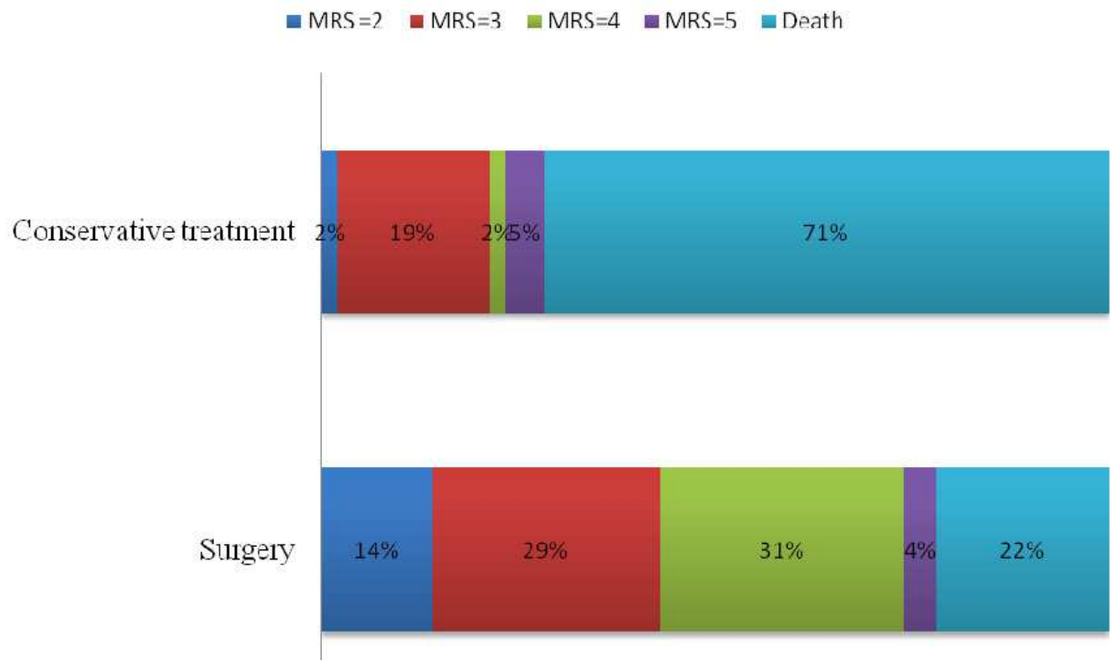


Table 4. Pooled analysis of DECIMAL, DESTINY, HAMLET, Distribution of the modified Rankin score after 12 months among the group treated with and without the decompressive surgery, (Vahedi et al,2007)

4.1.1.2 Patients selection

On the basis of the above mentioned pooled analysis, eligible criteria have been determined for carrying out the decompressive craniectomy(Table No 5).(Vahedi et al,2007; European Stroke Organization (ESO), 2009)

Inclusion criteria	Main Exclusion criteria
age range 18-60 years	prestroke score on the MRS > =2
ischemic infarction in the territory of the	coma with two dilated pupils
MCA with a score on the National Institutes of Health stroke scale (NIHSS) >15	other serious illness
less than 45 hours from the symptoms onset to surgery	contralateral ischemia or other brain lession
decrease in the level of consciousness to a score 1 or greater on item 1a of the NIHSS	
CT evidence of at least 50% infraction in the MCA territory,or infarct (volume > 145 cm3 on diffusion-weighed MRI)	

Table 5. Inclusion and Exclusion criteria of DC for MCA stroke

4.1.1.3 Timinig of procedures

One of the most important factors which decide about a good result in operated patients with malignant MCA infarction is the right timing of the operation. The timing is closely related to monitoring the intracranial pressure (ICP) increase, cerebral perfusion pressure (CPP) dynamics, also to radiologic monitoring of the development of the malignant edema as well as monitoring the clinical state of the patient. ICP and CPP measuring requires invasive approach. Nowadays there is guideline for ICP monitoring only for Traumatic Brain Injury management. (Adelson et al, 2003)

We use repeated CT examinations for monitoring progression of brain edema and midline shift. Besides noninvasive monitoring of the midline shift it is also possible to use noninvasive transcranial duplex colour coded sonography (TCCS) examination. Gerriets et al demonstrated that TCCS monitoring of midline shift is a useful tool in management of critically ill patients who cannot undergo repeated CT scans. (Gerriets et al, 2001) It must be pointed out that there are no exact radiologic indicative criteria for performing the DC.

Mori et al proved that the benefit of the operation is bigger even before the herniation of the brain tissue. In his study he divided patients with ischemia bigger than 200cm3 into 3 groups, he treated the first group conservatively, the second group was treated before the herniation of the brain tissue, and in the third group he carried out decompressive craniotomy but after herniation. In his work he proved a statistically significant benefit assessed after 6 months from the stroke in the Glasgow outcome scale (GCO) and also in the Barthels scale for the benefit of the patients who underwent the timely operation. (Mori et al, 2004) On the other hand, there are other studies which do not confirm the effect of timely " preventive " operations and these studies encourage clinicians to be more conservative and to wait for the time of developing the mass effect and midline shift. (Uhl et al, 2004; Rabinstein et al, 2006)

Bar et al demonstrated in his studies that the size of ischemia does not have any influence on the favourable outcome (mRankine 0-4) assessed 3 months after the operation. He also did not identify the timing as an important variable affecting the outcome but in his work only a few patients were indicated early and this masked the benefit of early versus late surgery. (Bar et al, 2011). In the pooled analysis (DECIMAL, DESTINY HAMLET) no difference was found between patients treated on the first and the second day. (Vahedi et al 2007) Hamlet demonstrates no benefit of late surgery between 48-96 hours from the stroke onset between groups of patients who were operated on and those who were not operated on. (Hofmeijer et al ,2009)

In conclusion on basis of the literature we believe that probably early decompressive surgery (that means before herniation and poor clinical status) is more beneficial than late timing. In our opinion "preventive surgery "up to 24 hours from the operation in patients with the whole middle cerebral artery territory stroke means prevention from irreversible demage of the brain tissue.

4.1.1.4 Utilization of decompressive surgery for malignant MCA infarction

The number of patients with malignant MCA ischemia who fulfilled the new indicative criteria for DC is not clear. Even the number of all patients who are indicated for DC is not

clear. Hacke et al study, 1993, which is cited by the majority of authors, implies that the prevalence of patients with malignant ischemia is approximately 10% of all the patients who suffered the cerebral MCA ischemia. Unfortunately there is no known data of how many patients with malignant ischemic stroke undergo decompressive surgery generally. (Bar et al, 2011)

Bar et al analysed the retrospective occurrence of a malignant edema in all the patients with MCA ischemic stroke who were admitted in 2009 into the Comprehensive stroke centre in Ostrava, the Czech Republic. They identified that 22 (10%) out of 217 patients admitted for acute ischemic stroke in the anterior circulation had a malignant supratentorial infarction and five patients (2.3%) met the indication criteria for decompressive surgery. Seventeen patients did not meet the criteria because they were aged >60 years in all cases.(Bar et al 2011)

In spite of the clear guidelines from 2008, the utilization of decompressive surgery for stroke patients with malignant ischemia did not increase essentially. In the Czech republic the number of procedures increased from 39 in 2006 to 56 in 2009. We estimate that only about 10% of the patients who met the criteria underwent the surgery. (Bar et al 2011)

In the United States the rate of hemicranietomies increasing by 21% per year but the operation was done only in 426 patients during the period between 2005-2009. That means in fact only 0.072 % of the patients with an acute MCA ischemic stroke who were registered in the Premier database. (Adeyoe et al 2010) There are several factors explaining the poor utilization of decompressive surgery:

- the guideline are relatively new and they have not yet entered the consciousness of neuorologists and neurosurgeons.
- clinicians have not yet associated with the idea that the outcome mRankin=4 is favourable and therefore do not indicate the patients for surgery .
- doctors do not believe that patient with ischemia size 50% MCA territory on a CT or 145cm3 without any signs of an edema or without a mass effect and midline shift should be indicated for an operation. They do not believe that “preventive” surgery is useful for patient.

In conclusion it is necessary to state that in the period of the past 5 years there has been a rise in the number of patients who underwent the operation, unfortunately this number is insufficient not only for the prevalence of the malignant ischemia occurrence but also for the number of patients who fulfil the guidelines criteria for DC as well.

4.1.2 Decompressive craniectomy for space occupying cerebellar ischemic infarction

Suboccipital Decompressive craniectomy with or without resection of necrotic cerebellar tissue is generally accepted among clinicians as an effective and lifesaving treatment strategy for cerebellar infarction. In spite of the lack of evidence based medicine this procedure is accepted more than craniectomy in malignant supratentorial infarction. (Merenda & DeGeorgia 2010; Mathew et al ,1995; Racoet al, 2003) Doctors fear a rapid expansion of the cereberall forman magnum or cerebellar tentorial herniation which leads to the compression of the brainstem followed by death. Deterioration of consciousness and

sixth nerve palsy are the first signs of brainstem compression. Mortality in this case has been estimated as high as 80% (Kakar et al, 2009; Ganapathy et al, 2003;Chen et al, 1992) Progressive deterioration of consciousness , decerebral fits and CT demonstration of mass effects strongly support performing decompressive surgery (with or without ventriculostomy for treatment of hypdrocephalus). The presence of the brainstem infarction has been associated with a poor outcome and the brainstem infarction has been analysed as the only independent predictive factor which has been associated with the poor clinical outcome. (Merenda & DeGeorgia 2010; Pfefferkorn et al, 2009; Chen et al, 1992) The other predictors, such as age, bilateral cerebellar infarction, and the time to surgery have not been significantly related to the poor outcome. (Pfefferkorn et al, 2009) Ventriculostomy and decompressive surgery are considered treatment of choice for space-occupying cerebellar infarctions (Class III, Level C).(European Stroke Organization (ESO),2009) But there are no randomized clinical trials which would prove this recommendation. (Adams et al, 2007) Currently we found no level I or II evidence to support of surgical treatment of space occupying cerebellar infarction. Therefore there is no optimal surgery strategy which would help choose patients with the highest benefit of the operation. The prognosis among survivors can be very good, even in patients who are comatose before the surgery. There is uncertainty of the prognostic value of age and preoperative Glasgow Coma Scale and large prospective case series is warranted.

4.1.3 Decompressive surgery for subarachnoid hemorrhage (SAH) and spontaneus intracerebral hemorhage (ICH)

Decompressive Craniectomy for SAH with elevated ICP remains controversial. We can notice that the intracranial pressure could escalate in both groups of patients with intracerebral haematoma; with the mass effect and also in patients with only subarachnoid hemorrhage where intracranial hypertension develops on the basis of the generalised brain swelling. In case of the delayed ischemic deficit, intracranial hypertension can occur between 5th and 15th day from the SAH onset. A number of recent studies have explored the role of craniectomy in the setting of aneurysmal subarachnoid hemorrhage associated with the large intracerebral hemorrhage (Schimer et al, 2008; Smith et al, 2002; Güresir et al, 2009). The patients indication, and the timing of the operation were discussed in past two decades. Schirmer et al showed that even in case of SAH without a large intracranial hematoma, DC led to a significant reduction in mortality. He reached a better outcome in an early DC (up to 48 hours) than in patients who undertook the decompression later. (Schimer et al, 2007) Nevertheless other authors have not confirmed these results and have not found any significant difference in the final outcome between the groups of patients with the elevated intracranial pressure who undertook the decompression and who were treated conservatively. (Buschmann et al, 2007; D'Ambrosio et al, 2005) There are no data nowadays for any kind of guidelines for performing DC in this indication. In our opinion DC for SAH with or without intracerebral hematoma should be considered only as an option of the treatment of the elevated intracranial pressure in a patient after SAH with or without intracerebral hematoma.

According to the only one randomized large study of the surgical treatment of ICH only in patients with lobar hemorrhage within 1 cm of the surface standard craniotomy may be

considered(Class IIb). (Steiner et al 2006) Decompressive craniectomy together with the ICH evacuation is supposed to be a life- saving procedure due to the decreasing ICP level. Some studies suggest that decompressive craniectomy and ICH evacuation might improve mortality in selected groups of patients. (Ma et al, 2010; Green et al 2010) Larger, randomized studies are needed to verify this recommendation.

4.1.4 Decompressive craniectomy for traumatic brain Injury (TBI)

It is recognized and widely accepted, that uncontrolled intracranial hypertension is associated with worse ourcome after traumatic brain injury. There are several deterious mechanisms starting immediately after traumatic impact resulting in secondary brain injury. These mechanisms may cause disruption of cellular haemostasis that leads to vicious circle elevated ICP - cell death – more oedema – worse perfusion – further elevation of ICP. Decompressive craniectomy is believed to interrupt this circle by decreasing ICP, but it has to be done early a appropriately sized. Despite wide and frequent use, to date there is no class I evidence showing improved outcome following decompressive craniectomy after TBI. In past 15 years 4 publications of class II and 23 of class III with positive conclusions were published. (Kakar et al, 2009) The most promising study on this topic to be under way is the RESCUEicp comparing the efficacy of DC versus optimal medical management for refractory intracranial hypertension following TBI. (Hutchinson et RESCUEicp investigators, 2006)

Up to date there are no specific guidelines or protocols stating exactly when or in what circumstances DC is appropriate, but there are some recommendations:

- A Cochrane review (2006) recommended DC may be justified in some children with medically intractable ICP after head injury but concluded there was no evidence to support its routine use in adults. (Sahuquillo & Arikan, 2006)
- European Brain Injury Consortium recommend DC as an option for refractory intracranial hypertension in all ages. (Maas et al,1997)
- The North American Brain Trauma Foundation suggests DC may be the procedure of choice in the appropriate clinical context and also considering the use of DC in the first tier of TBI management. (Bullock et al, 2006)

Most definitions of decompressive craniectomy describe this procedure as an option for managing refractory intracranial hypertension. Attention is focused on ICP, that is measured and therapy is aimed to lower rised intracranial pressure. Animal models confirm that decompressive craniectomy improves cerebral compliance and reduces ICP. (Zweckberger et al 2003)

But there are opinions that intervention in situation of refractory IC hypertension is delayed, and as known from our daily practice, in many cases decompression under these circumstances is predetermined for failure. In other words, we are looking for warning signs, that would induce early effective therapy that would preserve excesive brain swelling and conus formation. Microdialysis as a functional measurment and MRI perfusion/diffusion imaging with prognosis of extent and localization of tissue at risk (penumbra) seem to be very promising and are still waiting for clear definition of their roles.

5. Key steps of decompressive craniectomy

Decompressive craniectomy describes the temporary removal of a portion of the skull for the relief of high intracranial pressure. This can be achieved by removal of the fronto-temporal-occipital bone over one or both cranial hemispheres or can involve a bifrontal craniectomy. (Schirmer et al , 2008) Most common unilateral hemicraniectomy is typicaly indicated for unilateral space occupaying lesion. The procedure is started typically with large question mark skin incision and then large craniectomy is performed. Jiang in his work describes standard size of craniectomy 15x12cm to be more effective compared to limited craniectomy (8x6cm) (Jiang et al,2005). The procedure aims to reduce compression of brain structures, especially brain stem by swollen brain. Techniques describing simple bone removal without dural opening are believed not to be sufficient. There is no universally stadardized performance of DC and techniques may vary according to institution traditions. Anyway it is widely accepted, that decompression must be spacious enough to avoid cerebral tissue squeezing against the edges of craniectomy. The size of craniectomy defect seems to be crucial. It is stressed to remove temporal squama to ensure temporal lobe decompression (to avoid uncal herniation). Another point of discussion is dural closeure. Some authors do not close dura at all, some use auto- or allogenous grafts to perform duraplasty to prevent CSF leak and make the preparation for cranioplasty easier. Most recent essential requirements for "standard surgical technique" were described in DC for TBI in ongoing study (RESCUEicp) as follows: (Hutchinson et al, 2006)

- Wide (≥12 cm in diameter) decompressive craniectomy (avoiding brain herniation, a. k. a. fungus cerebri).
- Opening the dura and leaving it open (with an option of duraplasty).
- Avoiding tight bandage or positioning patient head on the craniotomy side, after decompression.

It is also recommended, although not absolutely essential:

- For diffuse brain swelling to use a bifrontal decompressive craniectomy with bilateral U-shaped opening of the dura, based on the superior sagittal sinus and with ligation and division of the sinus and falx anteriorly for maximum decompression of the frontal regions. The frontal sinus, if inadvertently opened during craniectomy, should be cranialized (excision of posterior wall, stripping of mucosa and plugging of osteum with the pericranium and/or free muscle and/or tissue glue).
- For predominantly unilateral swelling with midline shift a–wide (≥12 cm in diameter) "trauma" craniectomy with temporal decompression on the side of the swelling.
- If it is not feasible to keep the existing ICP monitor in place during the operation, to replace the ICP monitor following craniectomy via separate burr hole / bolt, at least 3 cm away from the bony edge of craniectomy.
- Performing cranioplasty within 6 months following decompressive craniectomy.

6. Complications

The procedure itself seems to be relatively safe with low reported occurence of acute surgical complications. Morbidity and mortality are associated with late comlications

secondary to surgical decompression. Many of these complications arise from normal pathophysiological alterations in cerebral compliance after removal of large piece of skull. Among well recognized complications are progression of haemorrhagic contusion, external cerebral herniation, subdural hygroma, infection, hydrocephalus, syndrome of trephined and epilepsy. Margules et al, 2010)

Yang et al. reported 50% complication rate after decompressiove craniectomy with 25,9% of patients who developed more than one complication. (Yang et al, 2008) There seems to be an association between the severity of the initial injury measured by the Glasgow Comas Scale, and the outcome of decompressive craniectomy. Yang et al in his work found patients with worse GCS score had higher complication rate and worse prognosis.

Herniation of swollen brain through craniectomy defect may significantly worsen patient´s prognosis, as it may lead to laceration of brain tissue and damage to cortical veins. Post-craniectomy brain oedema may be a consequence of hyperperfusion syndrome of decompressed brain. In Yang series brain herniation over bony edges has been reported in 27,8% of patients. This complication is more pronounced when small-sized craniectomy was performed. Techniques minimizing risk of herniation include suffitiently large craniectomy, augmentative duraplasty to limit cephalocele (this technique also limits postoperative hygroma formation) and insertion of "vascular cushions" formed by absorbable sponge adjacent to large draining veins to reduce risk of venostasis.

Nowadays with routine intraoperative antibiotic prophylaxis the risk of infection complications after decompressive craniectomy should not be more than 3-7%.Syndrome of the trephined (also known as sinking skin flap syndrome) appears weeks of months after creniectomy. (Stiver et al, 2009) Symptoms include headache, dizziness, irritability, concentration difficulties, memory problems and mood disturbancies. Sometimes also motor deficit may develop. The mechanism underlying this condition is probably transmission of atmospheric pressure over the brain tissue that impairs cortical brain perfusion. For this reason early cranioplasty is the treatment option.

7. Outcome

Early surgery – up to 24- 48 hours, age bellow 60 (and 50 years) and clinical status were identified as predictors of a favourable outcome after decompressive surgery in acute supratentorial stroke. (Vahedi et al, 2007; Bar et al 2011; Gupta et al, 2004) Unfortunately, radiologic criteria (infarction volume threshold and midline shift size) of a good clinical outcome have not yet been defined (Schimer et al, 2008). Only in patients where the ischemia is bigger than 145cm3 according to DWI MR which are made within 14 hours from the start of the stroke, there are potential candidates for malignant ischemia formation (Oppenheim et al, 2000). That means that patients with the MCA or MCA and Internal Carotid Artery (ICA) occlusion where early recanalisation has not been carried out and the brain ischemia in the region of the whole territory of the middle cerebral artery developed are potentially threatened by malignant edema. Patients with midline shift more than 4 mm according to transcranial color-coded sonography at 24 hours from stroke onset reached poor outcome (Gerriets et al, 2001). There is no evidence that patients with the dominance of infarction have a poorer favourable outcome than patients with

nondominance supratentorial infarction. The dominance of infarction should not be evaluated as an exclusion criterion for selection of patients to DC. (Merenda & DeGeorgia, 2010)

Bar et al identified that the clinical status in NIHSS was significantly and independently associated with a poor outcome, which was confirmed in many studies. (Bar et all 2010, 2011;Vahedi et al 2007; Gupta et al , 2004) DC performed prior to the clinical signs of herniation is associated with a favourable clinical outcome.(Chen et al 2007, Oppenheim et al,2000) A weakness of the randomized trials is the lack of the data on older patients . These randomized studies were carried out in patients younger than 60 and therefore the DC for malignant supratentorial infarction is recommended only for this age group in the recent guidelines. There is also evidence that DC can be beneficial even in older patients. (Jüttler & Hacke, 2011)

We conclude that the most important positive predictors of favourable outcome after DC in acute supratentorial stroke are age , clinical status in NIHSS ,time to surgery up to 24 (48) hours. For other indications (space occupying cerebellar ischemic infarction, SAH, ICH and traumatic brain injury) the outcome predictors have not been determined yet.

8. Expert suggestion

The intracranial hypertension means a very serious complication of various diseases of the central nervous system. The conservative treatment of ICP such as the management of the airway, breathing and circulation (ABCs), osmotherapy, sedation, steroid, hyperventilation, and induced therapeutic hypothermia very often fails and mortality in conservatively treated patients reaches 80%. Decompressive craniectomy is a surgical therapeutic option for the treatment of a massive middle cerebral artery infarction, space occupying cerebellar infarction, lobar intracerebral hemorrhage, severe aneurysmal subarachnoid hemorrhage and traumatic brain injury. The strongest evidence of the effectiveness of the treatment is nowadays available in patients with a malignant suratentoriálním infarction. Decompressive craniectory should be performed within 48 hours from the ischemic stroke occurance in every patient younger than 60 with a severe deficit (NIHSS scale more than 15 points) and at least a minor consciousness deterioration (Class I, Level of evidence A). Decompressive craniectomy in other types of a stroke is still a controversial issue. It is the most accepted by doctors in cases of space-occupying cerebellar stroke where the guidelines for executing the performance of type Class II, level of evidence C are valid. In case of subarachnoid and intracerebral haemorrhages there are no particular guidelines and doctors approach this treatment based on their individual experience and decisions.

In traumatic brain injury decompressive craniectomy is believed to interrupt the vicious circle of secondary brain damage by decreasing ICP, but it has to be done early a appropriately sized. There is no class I evidence showing improved outcome following decompressive craniectomy after TBI to date. The most promising study on this topic to be under way is the RESCUEicp.

9. Explicative cases

Case report 1. Supratentorial malignant ischemic stroke



Fig.1



Fig.2



Fig3



Fig.4



Fig.5



Fig.6

Case report: Supratentorial malignant ischemic stroke. Female, 25 years old, was admitted to hospital for severe rightside hemiparesis, gaze palsy and aphasia. CT angiography ACM-M1 segment artery occlusion (fig 1) Mechanical recanalization (Wingspanstent) was done within 5th hours from the stroke onset with only partial recanalization. (Digital subraction on angiography – fig 2) The CT scan 24 hours after the stroke onset shows massive ischemia in MC A territory on the left side (fig 3). The CT scan just before surgery showed space occupying lession, midline shift and tenitorial herniation (fig 4,5). The patient was operated on in 48 hours after the stroke. Decompressive craniectomy shows fig 6. She final outcome in 12 months time after the stroke is mRankin3, but cortical blindness is present. In our opinion, the patient was indicated to surgery too late and after tentoral herniation

Case report 2. Space occupying cerebellar infarction



Fig.1

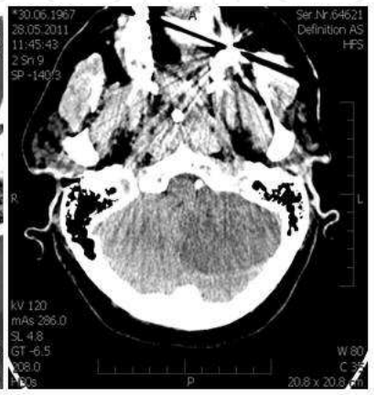


Fig.2



Fig.3



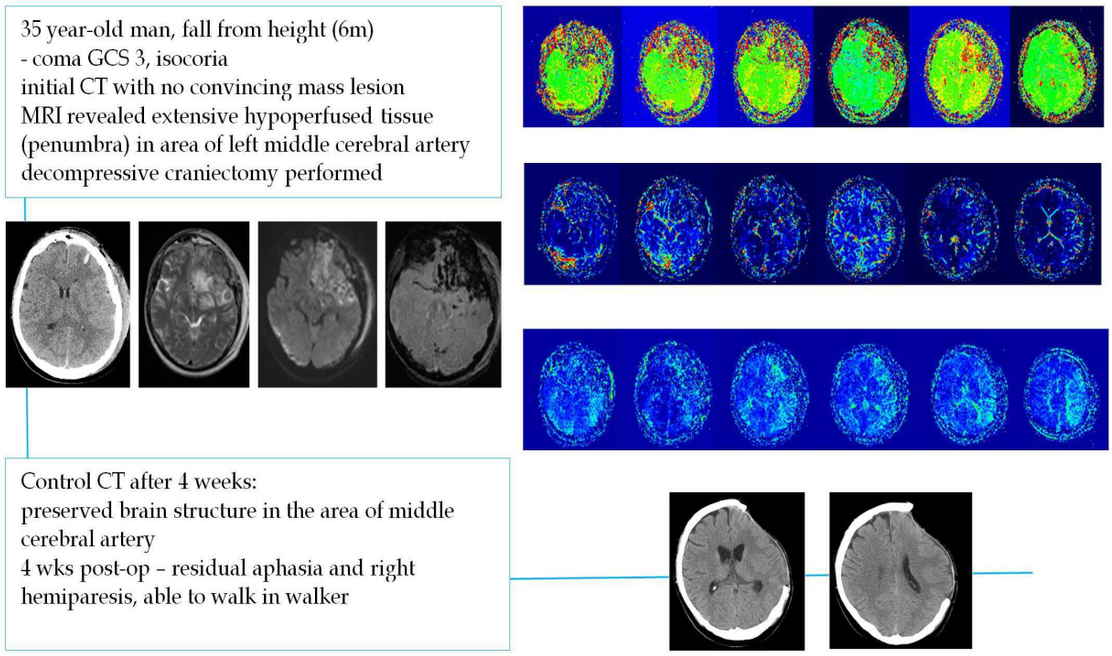
Fig.4



Fig.5

Case report: Space occupying cerebellar infarction. Male, 45 years old, was admitted to hospital for vertigo and desorientation. CT Angiography confirms occlusion of V4 segment of the right vertebral artery and the stenosis of V4 segment of the left vertebral artery (Fig 1). Mechanical recanalisation of the left vertebral artery was done within 5th hour, unfortunately iatrogenic occlusion of the left posterior inferior cerebellar artery (PICA) happened within the procedure (DSA, fig 4). This occlusion was followed by ischemia in PICA territory with the beginning expansion of the left cerebellar hemisphere and partial displacement of the 4th ventriculi (fig 2, 3). Suboccipital decompressive craniectomy with resection of necrotic tissue and duroplasy was done 72 hours after the stroke onsed (fig 5). The outcome in the modified Rankin score is 4 in three months after the stroke.

Case report 3. Traumatic brain injury



10. Conclusion

Decompressive craniectomy is widely used as the treatment of intractable intracranial hypertension in patients after severe traumatic brain injury and ischemic stroke. It is believed that sufficiently large and correctly performed craniectomy may significantly improve patients outcome. In our opinion „preventive decompressive surgery" up to 24 hours from stroke onset means prevention from irreversible damage of the brain tissue and can reduce disablity. But undisputably the most important factor that is still subject of discussion is the timing of such a radical surgical procedure, in order not only to reduce mortality but also to improve the quality of life of the patients.

In traumatic brain injury the timing of decompressive craniectomy seems to be crucial. Early selection of patients that would have benefit from decompression is chalenge for new diagnostic methods (brain tissue microdialysis and MRI perfusion/diffusion imaging).

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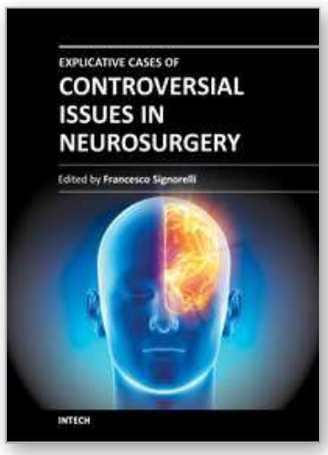
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