Decompressive Craniotomy in the Treatment of Malignant Ischemic Stroke of Cerebral Hemispheres (Review)

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The authors have considered the capabilities of decompressive craniotomy in malignant ischemic stroke treatment. Malignant stroke is understood to be stroke with extensive cerebral ischemia, post-ischemic edema formation, and the presence of lateral and/or axial cerebral displacement that is mainly due to proximal middle cerebral artery occlusion. There were highlighted the etiology and pathogenesis of this type of cerebrovascular disease. The disease predictors (clinical, radiological, laboratory) were described in detail, the most significant among them being emphasized. Based on randomized multicenter studies the researchers determined the indications, contraindications and selection criteria of patients when this surgical approach is used to treat malignant ischemic stroke. Surgical approach was demonstrated to enable to reduce case mortality, and improve functional treatment outcomes and results. There were described in detail the technique and peculiarities of decompressive craniotomy in this pathology, as well as the main postoperative complications. The authors considered the problem of optimal surgery time, and suggested an original treatment algorithm for patients with this pathology.

Key words: malignant ischemic stroke; cerebral decompression; hemicraniectomy; cerebral edema; NIHSS scale; DESTINY II; HAMLET.

Cerebrovascular diseases are still one of major medical and social problems of the contemporary world. In Russia, per year 400–450 thousand new cases of acute cerebrovascular disease (ACVD) are recorded [1–9], and worldwide over 7 million of ACVD are registered [10]. The mortality from strokes in developed countries ranks third among other diseases [2, 5, 10–14]. Ischemic strokes (IS) account for 70–85% of all ACVD [2, 3, 5, 10, 13, 15, 16]. About 31% patients after IS require continuous support, and 20% patients are not able to move by themselves [1, 3, 4, 6]. Only nearly 20% patients, who survived IS are able to return to previous job [1, 4, 6].

There is a special category of ACVD patients with the so called malignant ischemic stroke (MIS), which occurs in 10–15% cases [17–25]. MIS is understood as stroke with an extensive cerebral ischemic area with post-ischemic edema and lateral and/or axial brain dislocation [22, 25, 26]. MIS is usually related to the occluded proximal pat of medial cerebral artery (MCA) causing infarction in the area of over 50% of its blood supply [26, 27]. More frequently, MIS develops in young patients due to the absence of atrophy and lower brain tolerance. Patients with malignant course of the disease were found to be 10 years younger on the average than those with an ordinary IS course [18]. The mortality rate in MIS patients is 70–80%, and disability among survived patients is 80% [17–21, 23, 24, 26, 28–34]. Fatality in such patients is related to progressive brain edema, uncontrolled increase in intracranial pressure (ICP) and dislocated midbrain structures [24, 25, 34–37].

Malignant ischemic stroke pathogenesis

Blocked blood and oxygen entry the brain through a thrombosed vessel results in severe pathophysiological and pathobiochemical changes. Aerobic glycolysis is impaired leading to the glucose metabolism switch to an anaerobic process and, subsequently, resulting in lactate acidosis and accumulation of calcium ions. In turn, it is followed by the impaired functioning of ionic pumps, and chloride and sodium ions enter cells promoting cytotoxic edema development [1]. Edematous brain tissue causes local ICP. When local pressure is higher than the resistance of the surrounding brain matter, cerebral dislocation develops (Figure 1). Dislocation causes the displacement of midbrain structures resulting first to the compression of veins and local venous hypertension.
rise, and then to an additional local volume increase and obstruction of cerebrospinal fluid pathways resulting in sharp increase of ICP. Subsequently, it leads to artery compression and decreased perfusion of cerebral tissue, and not only in the infarction area. All that contributes to the infarction zone growth and progressive pathological process on a vicious circle principle [5, 26, 38]. Other satellite reactions are the following: impaired microvascular tone, edema of endothelial cells, activation of platelets, leukocytes and blood coagulation system [18]. Transtentorial herniation is the major reason for lethality in MIS. Case fatality within the first 48 h of the disease in such patients reaches 47%, and within 49–96 h the number increases 34%, i.e. total mortality in transtentorial herniation is 81% [26].

Prognostic factors of malignant ischemic stroke

Some authors have revealed direct relationship between ICP increase and the following transtentorial herniation development [29], moreover, brain dislocation develops earlier, if ICP rise is significant that accounts for patients’ physical condition aggravation [17]. Rapid augmentation of neurological signs, impaired consciousness level, a gaze deviation due to stroke, hemiplegia within the first 6 h after the disease onset combined with occluded proximal MCA are unfavorable prognostic factors [26].

Proved predictors of failure are the following: hypodensity of over 50% brain matter in MCA circulation, ischemic focus volume is over 145 cm³, lateral dislocation of midbrain structures is over 7–10 mm, the presence of axial dislocation of II and over degree according to Kornienko, hypodensity in frontal and parietal lobes is over 70%, and in temporal lobe it is over 80%, development of asymmetric obstructive hydrocephaly, reduced convexital cerebrospinal fluid space, smoothed cortical sulci, deformed lateral ventricles, compression of basal cisterns, cerebral perfusion is reduced by over 66%, the absence of agent accumulation in positron emission tomography in the area with volume of over 160 cm³ [18, 24, 26, 27, 29, 35, 39, 40–42].

Protein level of astrogliae S100B, which is released from cells after their death, is potentially useful laboratory technique for MIS diagnosis [26]. In stroke this protein specific for nervous tissue enters the blood reaching its maximum concentration level on days 2–4, and its amount depends on ischemic area extent. Infarction area volume is related to S100B level. The blood serum protein 24 h after the onset of the disease is 1.03 µg/L, and coupled with sensitivity (94%) and specificity (84%) can be a sign of massive cerebral edema [18]. Unfortunately, currently, the diagnostic technique is not available in any hospital, and its reliability in prognosis should be proved by larger studies.

Serena et al. [43] undertook a study to determine the concentration of molecular markers of blood-brain barrier (BBB) break in MIS patients. Within the first 24 h after the disease onset in blood plasma of patients they measured the levels of glutamate, TNF-alpha, glycine, IL-10, GABA, IL-6, matrix metalloproteinase-9 (MMP-9) and cell fibronectin (c-Fn). The patients with malignant ischemic stroke were found to have twofold MMP-9, and c-Fn level six times as high compared to the control group. The authors showed c-Fn level in serum equal 16.6 mg/ml (norm is 0.4 mg/ml) to be MIS predictor, the sensitivity being 90% and specificity about 100%.

Thus, astroglial protein S100B (>0.4 µg/L, norm <0.105 µg/L), matrix metalloproteinase-9 (>140 ng/mL, norm <25 ng/mL) and cell fibronectin (≥16.6 mg/ml, norm 0.4 mg/ml) can serve as molecular MIS markers.

Malignant ischemic stroke therapy

The position of decompressive craniotomy in malignant ischemic stroke therapy. In 1905 Cushing proposed a technique for decompressive craniotomy (DC) as a palliative technique in the treatment of brain tumors by reducing intracranial hypertension. First decompressive hemimicranionectomies in MIS were performed in 1930s [44, 45].
Table 1
Decompressive craniotomy results in malignant ischemic stroke

<table>
<thead>
<tr>
<th>Authors</th>
<th>Number of patients</th>
<th>Surivals</th>
<th>Deaths</th>
<th>Functional outcomes</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ivamoto et al., 1974 [46]</td>
<td>18</td>
<td>11</td>
<td>7</td>
<td>The authors draw attention to the when in addition to surgical decompression, dura opening, the female patient underwent the removal of necrotic cerebral tissue. During the postoperative period she could move by herself and do minimum household tasks 61% patients had unilateral mydriasis, 78% — impaired consciousness up to spoor or coma. Moreover, all patients were found to have hemiplegia or hemiparesis</td>
<td></td>
</tr>
<tr>
<td>Kondziolka, Falz, 1988 [47]</td>
<td>5</td>
<td>5</td>
<td>0</td>
<td>All patients could move by themselves postoperatively. Two patients managed to return to their previous job</td>
<td>The authors recommend surgery in case of mydriasis</td>
</tr>
<tr>
<td>Gupta et al., 2004 [48]</td>
<td>138</td>
<td>105</td>
<td>33</td>
<td>4 months after surgery 10 patients (7%) could look after themselves, 48 (35%) had slight or moderate neurologic impairment, and 80 (58%) patients died or suffered from severe neurologic impairment</td>
<td>All patients could move by themselves postoperatively. Two patients managed to return to their previous job</td>
</tr>
<tr>
<td>Curry et al., 2005 [49]</td>
<td>38</td>
<td>32</td>
<td>6</td>
<td>All survived had minimal neurologic impairment, and could moved by themselves</td>
<td>Authors state that patient’s age is one of the main prognostic signs of favorable outcome and improved functional outcome</td>
</tr>
<tr>
<td>Swiat et al., 2010 [50]</td>
<td>4</td>
<td>2</td>
<td>2</td>
<td>12 months after surgery two survived patients had severe neurologic impairment (4 points according to Rankine scale)</td>
<td>Authors call in question the technique of malignant ischemic stroke treatment</td>
</tr>
<tr>
<td>Krylov et al., 2013 [51]</td>
<td>13</td>
<td>7</td>
<td>6</td>
<td>— — — — — — — — — — — — —</td>
<td>— — — — — — — — — — — — —</td>
</tr>
<tr>
<td>Suyama et al., 2014 [52]</td>
<td>353</td>
<td>194</td>
<td>159</td>
<td>According to Rankine scale, 3 months after surgery, 5.2% patients had a favorable outcome (3 points and less), 22.9% patients had 4 points, 26.9% patients had 5 points (among those who survived)</td>
<td>Mean age of operated patients was 67, the patients over account for 80.2%. the authors concluded that patients’ age has no effect on survival rate and functional outcome</td>
</tr>
</tbody>
</table>

Table 1 shows in detail the results obtained using the technique [46–52]. Further, there were carried out a great number of studies comparing the results of surgical and conservative treatment of MIS. Many studies have shown the advantage of surgical management comparing surgical and conservative treatment of MIS. Table 2 demonstrates all research findings comparing surgical and conservative treatment of MIS. The problem of optimal surgery time in patients with the pathology is still unstudied. Some surgeons suggest an operation at an ultra-early stage, before edema and cerebral dislocation develop, while others prefer a delayed surgery, when edema and brain dislocation develop. Table 3 represents the findings of researches determining favorable surgery time (ultra-early or delayed) [60, 61]. There are a number of animal studies proving DC efficiency in MIS [62–66]. Thus according to the majority of studies, when DC is performed in patients with MIS, the lethality reduces significantly, and functional outcomes improve, especially in young patients [22, 23, 28, 32–34, 39, 67–82].

Enrollment criteria of patients

An advisory protocol of stroke management published in 2008 specifies the following criteria for patients to be enrolled in a group to perform DC:

1. age 18–60;
2. MCA infarction signs on CT 50% or more, or > 145 cm³ according to MR and/or DWI;
3. NIHSS is > 15 score;
4. depression of consciousness by 1 point and more according to 1a level of consciousness and lower;
5. surgery not later than 48 h since the onset of the disease.

One of the main success factors of MIS surgical treatment is a surgery time. Operative therapy should be preventive and ahead of the dislocation syndrome [18, 29]. A surgery cannot resuscitate "dead" neurons. The efficiency is possible in case the onset of the disease is possible in case the surgery is performed within the first 48 h after the symptoms appear [17]. An ultra-early surgery within 96-hour period (within the first 6 h after symptoms appear) produces the best results [18, 40, 75]. However, the efficiency is possible in case the surgery is performed within the first 48 h after the symptoms appear but the onset of the disease is delayed [16, 29]. A surgery not later than 48 h since the onset of the disease.

1) age 18–60;
2) MCA infarction signs on CT 50% or more, or > 145 cm³ according to MR and/or DWI;
3) NIHSS is > 15 score;
4) depression of consciousness by 1 point and more according to 1a level of consciousness and lower;
5) surgery not later than 48 h since the onset of the disease.
<table>
<thead>
<tr>
<th>Authors</th>
<th>Surgical treatment</th>
<th>Conservative treatment</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of patients</td>
<td>Survivals</td>
<td>Deaths</td>
</tr>
<tr>
<td>Holtkamp et al., 2001 [53]</td>
<td>12</td>
<td>8 (67%)</td>
<td>4 (33%)</td>
</tr>
<tr>
<td>DESTINY, 2007 [54]</td>
<td>17</td>
<td>14 (82%)</td>
<td>3 (18%)</td>
</tr>
<tr>
<td>HAMLET, 2009 [55, 56]</td>
<td>32</td>
<td>25 (78%)</td>
<td>7 (22%)</td>
</tr>
<tr>
<td>Integrative analysis (DECIMAL, DESTINY, HAMLET), 2009, 2013 [24, 56]</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Cho S.Y. et al., 2011 [35]</td>
<td>58</td>
<td>30 (51.7%)</td>
<td>28 (48.3%)</td>
</tr>
<tr>
<td>DESTINY II, 2011, 2014 [57, 58]</td>
<td>40</td>
<td>23 (57%)</td>
<td>17 (43%)</td>
</tr>
<tr>
<td>Raffiq et al., 2014 [59]</td>
<td>90</td>
<td>63 (70%)</td>
<td>27 (30%)</td>
</tr>
</tbody>
</table>
Table 3
Comparison of surgical (ultra-early and delayed) treatment results of malignant ischemic stroke

<table>
<thead>
<tr>
<th>Authors</th>
<th>Ultra-early surgery</th>
<th>Delayed operative intervention</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of patients</td>
<td>Survivals</td>
<td>Deaths</td>
</tr>
<tr>
<td>Schwab S. et al., 1998</td>
<td>31 (on average,</td>
<td>26 (84%)</td>
<td>5 (16%)</td>
</tr>
<tr>
<td></td>
<td>surgery was</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>performed 21 h</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>since the onset of</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>the disease)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cho D.Y. et al., 2003</td>
<td>12 (within 6 h</td>
<td>11 (92.7%)</td>
<td>1 (8.3%)</td>
</tr>
<tr>
<td></td>
<td>after symptoms</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>appeared)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

greater effect [61]. Therefore, all patients with MIS and the disease period less than 48 h should be examined by a surgeon to assess the operative treatment capabilities [18]. The age of 18–60 years is prognostically more favorable, but the efficiency of surgical treatment has been also proved in patients over 60 [19, 52, 84]. The dislocation of midbrain structures by over 7–10 mm is a surgical indication as well [19, 27, 75]. After systemic fibrinolysis, an operative treatment is also effective and does not differ in long-term postoperative functional outcomes [85]. DC is recommended in unilateral mydriasis or progressive consciousness level reduction [26].

DC is inadvisable if stroke is beyond MCA circulation, the disease duration is over 48 h, and the consciousness level is up to deep coma (less than 6 points) with bilateral mydriasis, in continuous stem symptoms, systemic blood disorders and severe somatic comorbidities [17, 18, 26, 32, 40]. The question of performing DC in MIS is to be decided in each specific case based on the wish of a patient or his family, as well as considering possible functional outcomes. Patient’s age and comorbidity presence are of importance when choosing surgery as a primary operative treatment. The model of MIS patients’ selection for DC looks as follows (Figure 2).

**Operative technique.** The operative intervention consists in the resection of a fronto-parieto-temporal fragment, opening and free flap plasty of dura mater [17, 26]. Trephine window should be not less than 12 cm in diameter, since the smaller size results in brain matter entrapment that reduces the survival rate from 80 to 55% [86]. So, in prospective studies (DESTINY I, DECIMAL,
HAMLET) the size of trephine window is not less than 12 cm in diameter.

The study by Chung [87] has demonstrated the maximal decompression size (over 14–16 cm in diameter, or over 399 cm²) compared to a large size (12 cm or 308 cm²) to increase the percentage of a successful outcome (according to Rankine scale <3) 3 months after stroke and reduce the number of poor results and postoperative lethality.

One of the skin incision types in DC is the incision of soft tissues in fronto-parieto-temporal region in the form of “a question mark” with the base situated near the tragus (Figure 3).

Another incision type is an incision in the form of a large “horseshoe” (Figure 4). This incision type is smaller, causes less bleeding, and enables to preserve the main trunks of superficial temporal artery, and soft tissue stage of the surgery takes less time [29].

The bone resection borders are the following: anterior (frontal) — along the pupillary line; posterior — 4 cm posteriorly from the external acoustic meatus; upper — up to the level of the superior sagittal sinus; lower — the bottom of middle cranial fossa (Figure 5). It is of primary importance to resect temporal squamosa in the basal direction, since at this level pacchionian foramen is located, where the brain stem is trapped in temporo-tentorial dislocation [17, 26, 88].

Craniotomy is followed by arciform incision of dura mater along the bone window perimeter, with it suturing to the periosteum for the purpose of epidural hematoma prevention [17, 26, 88]. Ischemic cerebral tissue is not resected [17, 26]. Further, free duraplasty is performed using a graft (synthetic, as a rule), which is fixed in several points at the top of the wound to prevent the transplant dislocation. Soft tissues should be sutured in layers and particularly carefully to provide...
wound integrity and prevent postoperative wound liquorrhea [17]. Some researchers combine DC and uncoparahippocampectomy, partial lobectomy, removal of necrotic brain matter and ICP sensor installation [26, 29, 49].

The most optimal time for subsequent cranioplasty is 1.5–6 months after DC [17, 18, 69]. In addition, the number of complications reaches 26%: epileptic attacks 15.6%, superficial wound infections 3.1%, hydrocephaly 3.1%, transient neurological impairments 3.1%, and osteomyelitis 2.1% [90]. Wachter et al. [91] reported the complication rate in 30% cases: 17.4% — aseptic osteonecrosis, 5% — wound infections, 2.5% — hematomas, 2.7% — hygromas, and 1.7% — poor cosmetic results.

The effect of decompressive craniotomy on treatment outcomes. Many authors have reported DC to have higher efficiency in MIS than that in severe craniocerebral injuries [69, 92]. It is related to the fact that the prolapse of cerebral matter in trephining defect is less due to decreased arterial inflow and less mass-effect due to unilateral impairment, whereas craniocerebral trauma is accompanied by diffuse cerebral edema [13]. MIS patients after DC are found to have ICP reduction, improved cerebral hemodynamics proved by CT-perfusion findings [29, 68, 93–96].

Unfavorable factors for cerebral hemodynamics are the following: surgery performed more than 48 h after the disease onset, over 10 mm displacement of midbrain structures, and patient’s age over 55 [68]. Currently, there are no eloquent evidences on ICP monitoring required in patients after DC [29, 97].

Unfavorable outcome factors in DC in MIS patients are considered to be the following ones: over 10 mm displacement of midbrain structures, age over 60, anisocoria, impaired consciousness level up to coma. In a number of cases, the factor is the infarction of all the blood supply territory of the internal carotid artery [29]. The effect of the anterior and posterior cerebral arteries involved in the infarction on the disease outcome has not yet been proved in operated patients.

The main DC complications in MIS are the following: postoperative epidural hematoma, meningitis, wound infection, parenchymatous brain injury, liquorrhea, hygroma, hemorrhagic conversion of the infarction zone, cosmetic defects [24, 89, 98]. All these complications deteriorate MIC course and prognosis, and in some cases can determine the severity of an underlying disease. So, a bone flap removal in the postoperative period results in trephined skull syndrome or “sunken skin flap” syndrome that causes the loss of life quality, cosmetic defects and disability percentage increase [99].

The main causes of death of such patients are the following: pneumonia, deep venous thrombosis of lower extremities, thromboembolia of the pulmonary artery, sepsis, multiple-organ-failure syndrome [89, 100].

Conclusion

The analysis of DC results in MIS enable to make convincing findings of reduced case mortality and improved functional outcomes in DC that offers an opportunity to advise the technique to be used in certain patients. This applies especially the patients under 60, who can be operated on early (less than 48 h since the onset of the disease), however, the death rate and severe disability have been determined to reduce among older patients. When performing DC, a correct technique of the surgery is of great concern: a trephine window being not less than 12 cm, resection of basal areas of the temporal bone, wide opening of dura followed by free flap plasty. Moreover, there should be kept in mind that the surgery itself bears a certain risk of complications: infectious, hemorrhagic, cosmetic.

The subject requires further investigations, which should be aimed at revealing MIS predictors, subsequent correction of indications and surgical contraindications, and determination of optimal time and volume of the operation.

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