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Hypothermia and Decompressive Surgery

Moon Ku Han, MD, PhD
Neurocritical Care Unit, Department of Neurology, Seoul National University Bundang Hospital, Seongnam, Korea

Decompressive surgery

The concept of decompressive surgery for treatment of elevated intracranial pressure the context of ischemic brain edema had been reported already in 1956. The rationale of this treatment modality consists of opening of the skull and removal of a bone flap to allow the edematous brain to swell outward, thereby preventing intracranial tissue shifts and life-threatening downward herniation. The most recent meta-analysis of DECIMAL, DESTINY, and HAMLET included all patients from the 3 trials who were randomized within 48 hours after symptom onset (n=109) and focused on mortality and functional outcome after 1 year. The average ages of enrolled patients are less than 60.

The absolute risk reduction for mortality comprised 49.9% (95% CI, 33.9 to 65.9), corresponding to a number needed to treat of 2 for prevention of death (Fig. 1). There was also a significant absolute risk reduction of 41.9% (95% CI, 25.2 to 58.6) with hemicraniectomy for a modified Rankin scale (mRS) 〉4 with a number needed to treat=2. Surgery, however, did not lead to a significant benefit in functional outcome when dichotomization between a mRS of 0 to 3 and 4 to 6 after 12 months was chosen (absolute risk reduction, 16.3%; 95% CI, -0.1 to 33.1). Reduction in mortality with surgical treatment was accompanied by an increase in moderate severe disability (mRS of 4) in survivors. Decompressive hemicraniectomy is a life-saving surgery that appears to benefit younger patients

Decompressive hemicraniectomy (DH) for malignant cerebral edema has been reported to decrease mortality rate, but this benefit has not been demonstrated in elderly patients.

Moon Ku Han, MD, PhD
Neurocritical Care Unit, Department of Neurology, Seoul National University Bundang Hospital, 300 gumidong, Bundang-gu, Seongnam City, Korea
Tel: +82-31-787-7469, FAX: +82-31-787-4059
E-mail: mkhan@snu.ac.kr

Therapeutic hypothermia

Therapeutic hypothermia, recently renamed targeted temperature management, including prophylactic normothermia, has been used to improve this secondary impact onto brain and other organ tissue. Therapeutic hypothermia/targeted temperature management aims to attenuate a cascade of secondary injury mechanisms, which is started immediately after the initial event (primary injury) and may last for hours and even days. It is now fully accepted and of a high level of evidenced medicine that in cerebral hypoxia (in a patient with cardiac arrest due to a shockable arrhythmia) as well as asphyxial encephalopathy a 24-hour therapeutic hypothermia (33 to 34°C), irrespective of the type of cooling, improves neurological outcome; that is, morbidity but also mortality. It needs to be stressed that even such seemingly similar diseases as global hypoxia (in cardiac arrest due to a shockable arrhythmia), asphyxial

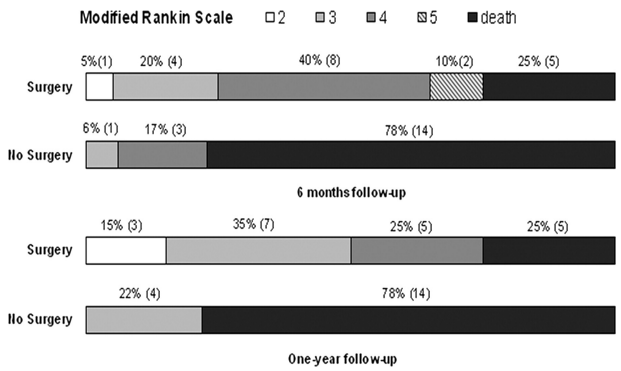

Figure 1. mRS score distribution in the 2 therapeutic groups at the 6-month and 1-year follow-up.

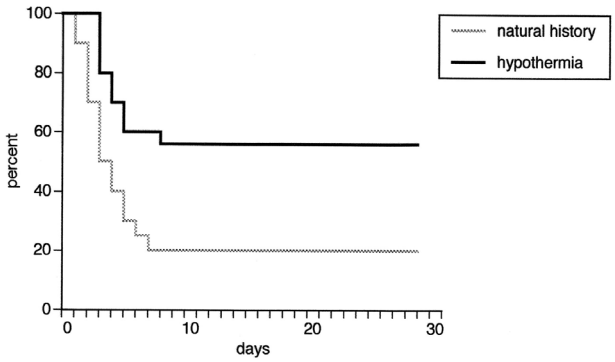

Figure 2. Survival curve of all patients treated with moderate hypothermia compared with patients treated with conventional therapy.

encephalopathy and ischemic stroke have so few pathophysiologic cascades in common. Therefore, they may not be treated all alike, in particular, with respect to type, duration, speed and depth of hypothermia as well as rewarming management.

It has already been demonstrated that in hypoxic encephalopathy hypothermia for 24 hours may be sufficient. However, disease entities such as ischemic stroke, hemorrhagic stroke with formation of peri-hematomal edema, the wide range of neuronal injuries after subarachnoid hemorrhage may present even more complex pathophysiologic mechanisms.

Any type of therapeutic measure, still being the subject of research, must never harm the patient.

In "malignant" MCA infarction, outcome is fatal in most patients, with a mortality rate of about 80% with standard treatment. In therapeutic hypothermia, the mortality rate was only 44%, and survivors reached a favorable outcome with a mean BI of 70 (Fig. 2). Preliminary results suggest a beneficial effect of moderate hypothermia can help to control critically elevated ICP values in severe space-occupying edema after MCA stroke and may improve clinical outcome.

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