Decompressive Hemicraniectomy After Aneurysmal Subarachnoid Hemorrhage

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Key words
- Cerebral aneurysm
- Decompressive hemicraniectomy
- Intracerebral hemorrhage
- Subarachnoid hemorrhage
- Vasospasm

Abbreviations and Acronyms
CPP: Cerebral perfusion pressure
CT: Computed tomography
DHC: Decompressive hemicraniectomy
HH: Hunt and Hess
ICP: Intracranial pressure
MAP: Mean arterial pressure
MCA: Middle cerebral artery
mRS: Modified Rankin scale
SAH: Subarachnoid hemorrhage
SCA: Superficial cerebellar artery
SD: Standard deviation
VSP: Vasospasm

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BACKGROUND: **The aim of this study was to document the effects of decompressive hemicraniectomy (DHC) on neurologic outcome in patients treated for aneurysmal subarachnoid hemorrhage (SAH) and developing otherwise uncontrollable intracranial hypertension.**

METHODS: **Sixty-six of the 964 patients (6.8%) treated for aneurysmal SAH underwent DHC and were stratified as follows: Group 1, patients undergoing aneurysm clipping and DHC in one surgical sitting (i.e., primary DHC). Group 2, patients receiving aneurysm embolization and thereafter undergoing DHC. Group 3, patients undergoing standard aneurysm surgery and requiring DHC later in the post-SAH period. Group 4, patients with insufficient primary DHC and later requiring surgical enlargement of the craniectomy.**

RESULTS: **Outcome was not influenced by the timing of DHC, but depended on the pathology underlying intracranial hypertension (i.e., whether lesions were primary hemorrhagic or secondary ischemic in origin). Patients with large hematomas, undergoing primary, secondary, or repeat DHC (46/66) had significantly better outcomes than the 20 patients treated for edema and delayed ischemic infarctions. There were 16 (34.8%) of the 46 patients in the hematoma group, but only 2 (10.0%) of the 20 patients in the ischemia group had favorable neurologic outcomes, defined as modified Rankin Scale scores 0–3 (*P* value = 0.038).**

CONCLUSIONS: **In the largest series of SAH patients to date who received both microsurgical and endovascular treatment of ruptured aneurysms, and who underwent DHC for otherwise uncontrollable intracranial hypertension. Neurologic outcome was significantly correlated with the pathology underlying intracranial hypertension. DHC beneficially affected neurologic outcomes in patients with space-occupying hematomas, whereas patients suffering delayed ischemic strokes did not benefit to the same extent.**

INTRODUCTION

In the acute phase after aneurysmal subarachnoid hemorrhage (SAH), uncontrollable intracranial hypertension can result in compromised cerebral perfusion and can lead to severe neurologic disabilities. Similar to embolic middle cerebral artery (MCA) infarction, where recent studies have demonstrated impressive benefits for patients undergoing decompressive hemicraniectomy (DHC) (3, 16, 20, 37, 38), patients suffering from intractable intracranial hypertension after aneurysmal SAH (2, 6, 8, 25, 33, 34) could also benefit from this neurosurgical procedure. Whereas recent reports have indicated that distinct subgroups might benefit from DHC after SAH (6), the role of DHC in this population is still in a process of being defined due to a paucity of experience. Moreover, the role of DHC in patients undergoing endovascular treatment of their ruptured aneurysms has not been systematically addressed in the literature. Therefore, the aim of this study was to assess neurologic outcome in the largest series of patients to date who are undergoing both microsurgical and endovascular therapy of acutely ruptured aneurysms, who later required DHC for treatment of otherwise uncontrollable intracranial hypertension.

PATIENTS AND METHODS

Since August 1993, there have been 964 patients treated for ruptured cerebral aneurysms from the Department of Neurosurgery at the Medical University of Vienna. Patient data were entered into a computer database, which were available for retrospective analysis. The present study is based on 66 patients of those 964 who were treated and met the following criteria: 1) SAH confirmed by head computed tomography (CT), 2) cerebral angiogram demonstrating intracranial aneurysms, 3) treatment of the ruptured aneurysm by either microsurgical clipping or endovascular coil embolization within 7 days of the most recent bleed, and 4) intractable intracranial hypertension requiring DHC.

Study Population and Treatment Protocol

The age of the patients ranged from 18 years to 70 years (mean, 51.0 years). The female to

Table 1. Patients Characteristics

	Total Study Population	Treatment Group 1	Treatment Group 2	Treatment Group 3	Treatment Group 4
Patient Number (n [%])	66	22 (33.3)	13 (19.7)	17 (25.8)	14 (21.2)
Patient Age (years)	50.3	50.7	47.7	52.1	49.9
Hunt and Hess Grades (n [%])					
5	19 (29)	10 (45)	3 (23)	4 (24)	2 (14)
4	25 (38)	8 (36)	8 (62)	5 (29)	4 (28)
3	16 (24)	4 (18)	1 (8)	6 (35)	5 (36)
2	3 (5)	0	1 (8)	1 (6)	1 (7)
1	3 (5)	0	0	1 (6)	2 (14)
Fisher Grades (n [%])					
4	47 (71)	22 (100)	6 (46)	11 (65)	8 (62)
3	16 (24)	0	6 (46)	5 (35)	5 (31)
2	3 (5)	0	1 (8)	1 (6)	1 (7)
1	0	0	0	0	0

Patients undergoing decompressive hemicraniectomy (DHC) in this series were stratified to the following treatment groups: Treatment group 1, patients undergoing aneurysm clipping and DCH in one surgical sitting (i.e., primary DHC). Treatment group 2, patients receiving endovascular aneurysm therapy and undergoing DHC either immediately before or after coil embolization, or in a delayed fashion. Treatment group 3, patients undergoing standard aneurysm surgery and requiring DHC later in the post-SAH period. Treatment group 4, patients with insufficient primary DHC and later requiring augmentation of the craniectomy.

male ratio was 3.2 to 1. Their clinical grades at admission were stratified according to the classification of Hunt and Hess (HH) (18). The amount of blood detected on the initial CT scan was scored according to the classification of Fisher (10). Details are given in **Table 1**.

Patients in poor admission HH grades who had signs of severe brain swelling detected intraoperatively or postoperatively by CT scans remained sedated after surgical and endovascular treatment (1, 22). Intracranial pressure (ICP) measurements were obtained in all patients. ICP was monitored by ventriculostomy (Integra Neurosciences Implants, Sophia Antipolis Cedex, France) and/or fiberoptic intraparenchymal monitoring systems (Camino, San Diego, CA). Mean arterial blood pressure (MAP) was continuously monitored and cerebral perfusion pressure (CPP) was calculated (CPP = MAP − ICP). Monitoring of cerebral blood flow velocity was performed in all patients using a 2 MHz range gated pulsed transcranial Doppler ultrasound (Multidop L, DWL, Germany). Because the measurement results of invasive neuromonitoring, including brain tissue oxygenation monitoring (39) and microdialysis (24) were not consistently available throughout the study period, these data were not included. Patients underwent a treatment protocol including calcium-channel antagonists (28) and hypertensive hypervolemic hemodilution (9, 36) was implemented in patients meeting defined criteria of posthemorraghic vasospasm (VSP) (12-14). Patients with cerebral blood flow velocity values > 200 cm/sec and/or suffering delayed ischemic neurologic deficits were evaluated angiographically and subjected to intra-arterial pharmacologic spasmolysis (30), balloon angioplasty (15), or both.

Patients receiving DHC for intractable intracranial hypertension were stratified as follows. Group 1 patients undergoing primary DHC in presence of 1) large, space-occupying hematomas and brain swelling as detected on preoperative CT scans, and 2) otherwise uncontrollable brain swelling occurring intraoperatively. In the majority of these cases, preparations for a sufficiently large DHC were made beforehand, including modified patient positioning and skin incisions. However, patient positioning as required for adequate approaches to the target aneurysm still limited the average size of bony exposure available for DHC, resulting in slightly smaller craniectomies. In selected cases, in which significant brain swelling was unexpectedly encountered intraoperatively, the bone flap was not reinserted, the craniotomy was enlarged to the maximum possible extent, and an augmentational duraplasty was performed. Due to the limitations of the initial skin incision, in this selected subgroup primary DHC did not reach the sizes possible in patients with secondary DHC. Group 2 patients receiving endovascular treatment of their ruptured aneurysm and developing intractable intracranial hypertension immediately or in a delayed fashion. Because there were no limitations for skin incision and craniotomy, these patients usually received extensive DHC. Group 3 patients undergoing uneventful cerebral aneurysm surgery, but later developed intracranial hypertension. These patients had a second operation consisting of removal of the small pterional bone flap and extensive DHC. The pre-existing skin incision was usually extended to the occipito-parietal region in a "Y" shape, giving enough space for sufficient DHC. Group 4 patients initially stratified into group 1, who suffered intracranial hypertension despite primary DHC, underwent repeat DHC with surgical enlargement of the craniectomy.

For patients undergoing primary DHC, the necessity for surgical decompression is usually assessed: 1) preoperatively from CT imaging demonstrating space-occupying hematomas or significant brain swelling, or 2) intraoperatively by the neurosurgeon according to personal experience and judgement of the intraoperative situation. For patients stratified in groups 2 to 4, structured management algorithms providing ICP cut-off values have been reported (6), but were not used in this series for the following reasons: 1) neurologic deficits may occur in absence of relevant intracranial hypertension, with mass effect and brain shift from edema, infarction, and hemorrhage resulting in axial brain stem compression (7, 23); 2) the ICP value measured must be correlated to the treatment intensity required to correctly estimate the severity of intracranial spacial decompensation. Recently published ICP treatment scores have supported this notion (32). Because such scores were not calculated beforehand it was impossi-