Suboptimum hemicraniectomy as a cause of additional cerebral lesions in patients with malignant infarction of the middle cerebral artery

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Object. The goal of this study was to determine the frequency of hemicraniectomy-associated lesions and their potential effect on the risk of mortality in patients suffering from malignant infarction of the middle cerebral artery (MCA).

Methods. The authors evaluated serial computerized tomography scans obtained in 60 patients with complete infarction of the right MCA who were treated using hemicraniectomy. The maximum diameter of the hemicraniectomy was determined and the hemicraniectomy-associated lesions were classified as ischemic lesions or hemorrhages. The category of hemorrhages was composed of parenchymal, subdural, or epidural/subgaleal hematomas.

Parenchymal hemorrhages and infarcts associated with hemicraniectomy occurred with frequency rates of 41.6% and 28.4%, respectively. The occurrence of hemicraniectomy-associated bleeding was related to the size of the hemicraniectomy performed; that is, the smaller the hemicraniectomy, the more often lesions occurred (p < 0.05). Hemicraniectomy-associated bleeding was also related to an increased risk of mortality.

Conclusions. Hemicraniectomy is an effective therapy in patients with malignant infarction of the MCA. However, a hemicraniectomy that is too small in diameter may cause the formation of additional lesions and adversely affect the risk of mortality and the quality of survival. In addition, the size and shape of the edges of the bone defect are important factors relating to its efficacy.

KEY WORDS • cerebrovascular disease • stroke • hemicraniectomy

The majority of patients with complete infarction of the MCA experience space-occupying brain edema. In the absence of interventional therapy, 80% of these patients die of transtentorial herniation. A therapeutic approach that may reduce this mortality rate is hemicraniectomy. In recent studies it has been reported that hemicraniectomy may also be effective treatment for other diseases that carry the risk of brain swelling, such as traumatic brain injury. Hemicraniectomy follows the simple principle of creating space for swelling cerebral tissue by removing portions of the osseous skull and performing subsequent duraplasty. In theory, decompressive hemicraniectomy may also improve perfusion of collateral leptomeningeal vessels, improve retrograde perfusion of the MCA, optimize perfusion of the penumbra, and, consequently, reduce infarct size and neurological deficit. In a stroke model in rats, early craniectomy has been shown to result in significantly smaller ischemic lesions. Authors of a large controlled study involving patients with infarctions of the right MCA territory documented a 34% mortality rate in patients who underwent hemicraniectomy, compared with a 78% rate in those who did not.

Depending on the size of trephination, one may observe cerebral lesions that are independent of the initial infarction and are exclusively located next to the edge of the trephination. In addition, intracranial hemorrhages develop in some patients. In this paper we report on 60 patients with large cerebral MCA infarcts, who were prospectively treated using hemicraniectomy and later analyzed for the occurrence of hemicraniectomy-related lesions.

Clinical Material and Methods

Sixty patients (40 men and 20 women) with space-occupying MCA infarcts were treated with hemicraniectomy. The mean age of these patients was 50.4 ± 10.3 years at the time of surgery. At admission, each patient was evaluated using the Scandinavian Stroke Scale and the Glasgow Coma Scale. Computerized tomography scans were obtained on the day of admission and on Days 2 and 6 posthemicraniectomy.

Operative Procedure

The procedure for decompressive surgery has been described previously. Briefly, a large bone flap with a circular to oval shape (diameter 11–12 cm) is removed. The

Abbreviations used in this paper: CT = computerized tomography; MCA = middle cerebral artery.
fig. 1. Computerized tomography scan demonstrating a hemicraniectomy-associated hemorrhage in close apposition to the frontal bone edge. Note the sharp edge of the bone.

excision includes the frontal, temporal, parietal, and small portions of the occipital squamae. The dura is affixed to the edge of the craniotomy to prevent epidural bleeding. A biconvex dural patch made of lyophilized dura from a cadaver or homologous temporal fascia is then placed within the incision. The length of the dural patch is 18 to 20 cm and the width is 2.5 to 3.5 cm.

Determination of Hemicraniectomy-Associated Lesions

We analyzed serial CT scans to determine the occurrence of hemicraniectomy-associated infarcts and hemorrhages. Ischemic lesions associated with the hemicraniectomy were defined as those occurring close to the edges of the trephination and being clearly distinct from the initial site of cerebral ischemia. Hemorrhages were classified as caused by shearing if they were located at the edges of the bone defect, or as subdural or epidural/subgaleal hematomas. Some epidural/subgaleal hematomas are caused by a hemorrhagic temporal muscle. We also determined the size of the trephination by measuring its maximum diameter observed on the CT scan. In this series of patients, the shape of the removed bone flap always approximated that of a circle; hence, this rather simple measurement (diameter) of trephination size should entail minimal loss of information. The shape of the bone edges (flattened or sharp) was determined by morphological analysis. If a protruding edge or even the smallest spinelike structure that might represent bone or loose dura could be observed on the CT scan, the shape was defined as being sharp. Extensive beveling leads to profuse venous bleeding and, therefore, is not done in this operation.

Hemicraniectomy-associated lesions were also classified according to size: small (< 2 ml), medium (2–10 ml), and large (> 10 ml). For image analysis, tapes of the CT scans were analyzed using a commercially available computer software program (Voxel Q; Marconi Medical Systems, Cleveland, OH).

Statistical Analysis

Summary statistics are reported as the means ± standard deviations. Interrater reliability for the presence or absence of hemicraniectomy-associated lesions was assessed using kappa statistics. Relationships among the presence of hemicraniectomy-associated lesions, sharp bone edges, and clinical outcomes (measured by survival) were determined using chi-square tests. Relationships between the size of the trephination and the occurrence of hemicraniectomy-associated ischemic lesions or hemorrhages were tested using the Student t-test.

Results

Sixty patients suffering from acute stroke were examined using pre- and posthemicraniectomy CT scanning. On admission, in these patients the mean Scandinavian Stroke Scale score was 19 ± 8.4 (median 17) and the mean Glasgow Coma Scale score was 8 (range 4–13). All patients initially presented with a dense hemiparesis and forced eye and head deviation. In 42 patients there was infarction of the entire MCA territory. In nine patients additional infarcts were identified in the territory of the anterior or posterior cerebral artery. The survival rate at 30 days postadmission was 80% (48 of 60 patients). Interrater reliability for the presence or absence of hemicraniectomy-associated lesions was quite satisfactory (κ = 0.86).

Hemicraniectomy-associated infarcts or parenchymal hemorrhages of any size occurred in 70% of all patients. Hemicraniectomy-associated parenchymal hemorrhages (Fig. 1) and hemicraniectomy-associated infarcts occurred with a frequency of 41.6% and 28.4%, respectively. Most lesions were small (Table 1). Small epidural or subgaleal hemorrhages occurred in 10% of all patients. Small subdural hematomas were evident in three patients. Only one patient experienced a large epidural hematoma, which had to be revised operatively. In another patient, the trephination wound had to be revised because of signs of infection.

There was a significant association between the frequency of bleeding caused by hemicraniectomy and the size of the bone defect (p < 0.05). The mean size of the hemicraniectomies was 10.5 ± 2.8 cm. The smaller the craniectomy, the more often these lesions were encountered. No such relationship could be found for hemicraniectomy-associated infarcts or epidural hemorrhages. Another factor related to hemicraniectomy-associated lesions of any type was sharp bone defect edges (p < 0.05). The mortality rate was significantly higher (p < 0.05) in the group of patients with hemicraniectomy-associated pa-

TABLE 1

<table>
<thead>
<tr>
<th>Hemicraniectomy-Associated Lesions</th>
<th>No. of Patients (%)</th>
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<tbody>
<tr>
<td>ischemic infarct</td>
<td>13 (21.7)</td>
</tr>
<tr>
<td>parenchymal hematoma</td>
<td>17 (28.3)</td>
</tr>
<tr>
<td>epidural/subgaleal hematoma</td>
<td>6 (10)</td>
</tr>
<tr>
<td>subdural hematoma</td>
<td>3 (5.0)</td>
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</tbody>
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Suboptimal hemicraniectomy in patients with MCA infarction

Discussion

We retrospectively evaluated the CT scans of 60 patients with malignant infarcts of the MCA to determine the occurrence of hemicraniectomy-related lesions such as ischemic infarcts and parenchymal, epidural, subgaleal, and subdural hemorrhages. We found ischemic lesions or parenchymal hemorrhages in 70% of these patients. Small epidural or subgaleal hemorrhages were found in 10% of the patients. Hemicraniectomy-related bleeding was associated with an increased risk of mortality, and small bone defects were associated with hemicraniectomy-associated hemorrhages.

We chose the maximum diameter of the bone defect measured on the CT scan as the size of the hemicraniectomy in this limited study, because the removed bone flap in our patients invariably had an almost circular shape. Larger therapeutic trials are needed to determine the influence of herniating volume or, at least, the size of the craniectomy on the decompressing effect of the surgery. The described observation of sharp trephination edges on the CT scan is probably more often due to protrusion of a dura that is too loose than to sharp bone edges, because neurosurgeons typically take care to produce no sharp angles. The angle in our study was 90°.

In a model of focal cerebral ischemia in the rat, hemicraniectomy was shown to reduce the incidence of mortality and increase functional scores. In two prospective clinical series of patients with large hemispheric infarcts results have demonstrated that survival is significantly increased by craniectomy. We recognize the limitations inherent in the results of these studies, as well as those in our own findings: rather small patient cohorts, nonrandomized designs, and lack of blinding. Larger multicenter trials are needed to validate and further refine these results.

On the basis of our findings, we can only draw conclusions indirectly about the mechanisms that lead to the hemicraniectomy-associated lesions observed in our patient cohort. It is plausible, however, that shear forces at the edge of the trephination caused by a sudden loss of counterpressure on the part of the ipsilateral skull may lead to lesion formation. This would also explain the association of small bone defects with bleeding due to shearing. Smaller craniectomies do cause a higher shear stress on the swollen brain (Fig. 3). This is especially true if the edges of the craniectomy are sharp (Fig. 1). Alternatively, decompression of the brain may result in decreased interstitial fluid pressure and, thereby, increased edema, which again can lead to an increase in shear forces. In experimental craniectomy, a decrease in tissue pressure is significantly more pronounced in the cortex—where hemicraniectomy-associated lesions occur—than in the basal ganglia.

These mechanisms may well account for the observed ischemic lesions and parenchymal hemorrhages. Epidural hemorrhages are often caused by the wound surface and are sometimes indistinguishable from a hemorrhagic temporal muscle.

Small craniectomies with a diameter of 10 cm or less lead to an increased number of hemicraniectomy-associated hemorrhages. Although no direct relationship between the size of the bone defect and the rate of survival could be established in our study, small craniectomies are predisposed to shearing-associated bleeding, which in turn is related to a higher mortality rate. The findings in this cohort of patients with malignant infarction of the MCA are also likely to pertain to patients undergoing hemicraniectomy for different reasons, such as traumatic brain injury. To achieve an optimum decompressive effect, the size of the craniectomy is crucial. Doubling the diameter from 6 to 12 cm results in an increase in decompressive volume from 9 to 86 ml. The craniectomy should be performed in the frontotemporoparietal region and reach the base of the frontal bone. Removal of portions of the occipital squamae might be problematic and may cause problems of stability and patient positioning. This limits the size of the hemicraniectomy. The midline should be spared by 1 cm because opening of bridging veins will cause additional bleeding. For duraplasty the dura should be opened us-
ing one longitudinal and three radial incisions that almost reach the osseous rim; a large (2–3 cm) patch of replacement dura, made of lyophilized dura from a cadaver or homologous temporal fascia, should then be placed within the incision.

Conclusions
Craniecetomy-associated lesions occur in more than 50% of patients, probably due to an increase in shear forces acting on the brain. However, only localized hemicraniectomy-associated bleeding and a small craniectomy diameter correlate with an increased rate of mortality. If therapeutic craniectomy is performed, the size of the bone defect should be at least 12 cm and additional duraplasty should be performed.

Further studies that involve larger patient cohorts are needed for an accurate determination of the optimum size of craniectomies.

References

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