Sinking Skin Flap Syndrome and Paradoxical Herniation After Hemicraniectomy for Malignant Hemispheric Infarction

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Background and Purpose—“Sinking skin flap” (SSF) syndrome is a rare complication after large craniectomy that may progress to “paradoxical” herniation as a consequence of atmospheric pressure exceeding intracranial pressure. The prevalence and characteristics of SSF syndrome after hemicraniectomy for malignant infarction of the middle cerebral artery are not well known.

Methods—We analyzed a prospective cohort of 27 patients who underwent hemicraniectomy for malignant middle cerebral artery infarction. All had a clinical and brain imaging follow-up at 3 months and were followed until cranioplasty.

Results—Three of 27 patients (11%) had, at 3 to 5 months posthemicraniectomy, SSF syndrome with severe orthostatic headache as the main symptom. In addition, 4 patients (15%) had radiological SSF syndrome but no clinical symptoms except partial seizures in one. Patients with SSF syndrome had a smaller surface of craniectomy (76.2 cm² versus 88.7 cm², \(P=0.05\)) and a tendency toward larger infarct volume, an older age, and a longer delay to cranioplasty than those without this syndrome.

Conclusions—SSF syndrome either clinically symptomatic or asymptomatic affects one fourth of patients 3 to 5 months after hemicraniectomy for malignant middle cerebral artery infarction. It should be diagnosed as early as possible to avoid progression to a paradoxical herniation. (Stroke. 2010;41:560-562.)

Key Words: complications ■ surgery ■ hemicraniectomy ■ malignant cerebral artery infarction ■ herniation

The syndrome of the “trephined” or the “sinking skin flap” (SSF) syndrome is a rare complication after a large skull bone defect. It consists of a sunken skin above the bone defect with neurological symptoms such as severe headaches, mental changes, focal deficits, or seizures. The SSF may progress to “paradoxical herniation” as a consequence of the atmospheric pressure exceeding intracranial pressure and may eventually lead to coma and death.

The objective of our study was to determine, in a prospective cohort of malignant infarction of the middle cerebral artery (MCA), the prevalence and characteristics of SSF syndrome and of any radiological sunken skin flap without symptoms after hemicraniectomy.

Materials and Methods

Patients

All patients randomized in the surgical arm of DEcompressive Craniectomy In MALignant middle cerebral artery infarcts (DECIMAL), a trial that compared medical treatment and hemicraniectomy for malignant MCA infarction in our stroke center after the end of DECIMAL inclusion but according to the same criteria, were considered.

All surviving patients had at the 3-month follow-up a clinical and brain imaging evaluation (axial and/or coronal fluid-attenuated inversion recovery MRI and/or axial and/or coronal CT scan) and were then followed every 3 months for 1 year. The timing and procedure of cranioplasty were left to the discretion of the neurosurgeon. The study was approved by an institutional ethics committee, and the patient or a close relative gave informed consent.

Radiological Evaluation

The following measures were performed by the same neuroradiologist (J.-P.G.): (1) the maximum horizontal surface of the skull was estimated either on axial CT scan or axial fluid-attenuated inversion recovery MRI using the following formula: \(\pi/4 \times A \times B\) in which “A” was the maximum distance from the anterior and posterior inner tables of the skull and “B/2” the half-depth to the inner skull surface opposite to the bone flap measured at the midpoint of “A”; (2) the whole surface of craniectomy was estimated either on coronal reformation and axial plane CT scan or axial and coronal fluid-attenuated inversion recovery MRI using the following formula: \(\pi/4 \times C \times D\) in which “C” was the maximum vertical diameter and "D" the whole width of craniectomy.
“D” the maximum horizontal diameter of the bone defect; and (3) radiological SSF syndrome was defined by any anterior or posterior negative skin depression below a horizontal line drawn from both anterior and posterior outer tables of the bone defect at any of the following 2 levels: just above the plane of the thalamus (“thalamus” level) and just above the lateral ventricles (“ventricles” level). The measures were performed at 3 months and, in the case of delayed cranioplasty, at the last brain imaging when available.

**Statistics**

To evaluate the associations between SSF syndrome and baseline clinical and radiological parameters, we used a Fisher test for categorical variables and an analysis of variance for continuous variables.

**Results**

A total of 40 patients were considered. Among the 31 surviving patients, one was excluded because of brain abscess and 3 because of missing imaging data. Thus, a total of 27 patients (15 men, 12 women; mean ± SD age, 43.6 ± 9.3 years; range, 22 to 55 years) were analyzed.

During follow-up, 3 patients had SSF syndrome with severe orthostatic headache (Table 1). In one patient, orthostatic headache progressed in a few days to confusion, drowsiness, bilateral Babinski sign, subfalcine herniation, and midbrain compression (Figure). One additional patient had

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**Table 1. Patients’ Characteristics**

<table>
<thead>
<tr>
<th></th>
<th>Patients With SSF Syndrome</th>
<th>Patients With Asymptomatic Radiological SSF</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Patient 1</td>
<td>Patient 2</td>
</tr>
<tr>
<td>Gender/age, years</td>
<td>Male/47</td>
<td>Male/45</td>
</tr>
<tr>
<td>Infarction of the dominant hemisphere</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Baseline diffusion-weighted imaging infarct volume, cm³</td>
<td>220</td>
<td>300</td>
</tr>
<tr>
<td>Time from craniectomy to symptoms</td>
<td>5 months</td>
<td>4 months</td>
</tr>
<tr>
<td>Orthostatic symptoms</td>
<td>Headache, vomiting, drowsiness, bilateral Babinski</td>
<td>Headache, vomiting</td>
</tr>
<tr>
<td>Other symptoms</td>
<td>Partial motor seizures</td>
<td></td>
</tr>
<tr>
<td>SSF</td>
<td>Severe</td>
<td>Severe</td>
</tr>
<tr>
<td>Subfalcine herniation</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Midbrain compression</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Emergency treatment and response</td>
<td>Supine position, normal saline (9%)/complete resolution</td>
<td>Supine position, 1.5 L/day of oral hydration/partial resolution</td>
</tr>
</tbody>
</table>

**Figure.** Brain imaging of Patient 1: (A) axial diffusion-weighted imaging: right MCA infarction; (B) axial CT scan 24 hours later: large craniectomy with slight midline shift; (C–D) axial CT scan at 3 months: right hemispheric hypodensity at the thalamus level (D) and moderate anterior skin depression at the ventricles level (C); (E–G) axial CT scan at the time of clinical signs of brain herniation: sunken skin flap and severe compression of the right hemisphere with subfalcine herniation; (H) CT scan, 3-dimensional volume: profound sunken skin flap; (I) axial fluid-attenuated inversion recovery and (J) coronal T1-weighted image after 48 hours of supine position and intravenous hydration: complete resolution of the sunken skin flap.
radiological SSF and had 2 short episodes of partial seizures. Three other patients, all with severe global aphasia, had at 3 months radiological SSF but no orthostatic symptoms. None had a precipitating factor such as lumbar puncture.

Supine position with the head turned to the side of the craniectomy and intravenous fluid administration rapidly resolved orthostatic symptoms in 2 patients. One patient remained with a sunken skin flap for several weeks, because hemodynamic measures were not possible because of heart failure. However, after cranioplasty, the headaches completely resolved and the patient was able to move his hemiplegic arm against gravity, which was not possible before the operation.

When comparing patients without (n = 20) and those with SSF syndrome either clinically symptomatic or asymptomatic (n = 7), we found in patients with this syndrome a significantly smaller cranietomy surface (76.2 cm² versus 88.7 cm², P = 0.05) and a nonsignificant tendency toward a larger baseline infarct volume (SD), cm³ (87.0 versus 76.2, P = 0.29). In addition, smaller size of the craniectomy may lead to secondary injuries because of inadequate hemispheric decompression and finally to a larger hemispheric lesion. However, these findings have to be confirmed in a larger cohort of patients.

After hemisicraniectomy for malignant hemispheric infarction, the best time for cranioplasty is not well known. Considering our data, it may be justifiable to replace the bone defect during the first 2 to 3 months poststroke. In addition, SSF syndrome should also be considered as a delayed complication of hemisicraniectomy in the evaluation of its long-term benefit.

Acknowledgments
We thank all the DECIMAL investigators.

Sources of Funding
The DECIMAL trial was supported by grants from the Programme Hospitalier de Recherche Clinique of the French Ministry of Health. The study was sponsored by “Département de la Recherche Clinique et du Développement d’Assistance Publique–Hôpitaux de Paris” (AOM00148, P001004). M.S. received grants from the “Département de la Recherche Clinique et du Développement d’Assistance Publique–Hôpitaux de Paris” (AOM00148, P001004) and from “ARNEVA” (Association pour la Recherche en Neurologie Vasculaire).

Disclosures
None.

References
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*Stroke*. 2010;41:560-562; originally published online January 7, 2010;
doi: 10.1161/STROKEAHA.109.568543

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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