Epidemiology, diagnosis and treatment of cerebral venous thrombosis
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Decompressive hemicraniectomy in cerebral sinus thrombosis: consecutive case series and review of the literature

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Abstract

**Background and purpose:** Thirteen percent of patients with cerebral venous and sinus thrombosis (CVT) has a poor clinical outcome. In patients with a poor prognosis, endovascular thrombolysis can be considered, but this procedure does not appear to be beneficial in patients with impending transtentorial herniation due to large hemorrhagic venous infarcts. Therefore, halfway 2006, we changed our policy to decompressive hemicraniectomy in these patients.

**Methods and results:** Patients with CVT and impending herniation due to venous infarcts were eligible for surgical intervention. Since 2006 we consecutively treated 3 patients with decompressive hemicraniectomy. Two patients had an excellent outcome. The third patient, who had been comatose for at least twelve hours prior to surgery, died despite intervention.

**Conclusions:** Our data suggest that decompressive hemicraniectomy can be life-saving and can result in an excellent outcome in patients with severe CVT.
Case series of decompressive hemicraniectomy in CVT

Introduction

Approximately 13% of patients with cerebral venous and sinus thrombosis (CVT) has a poor clinical outcome.\(^1\) Transtentorial herniation due to mass lesions is the most common cause of death. The International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT) identified risk factors that predict poor outcome, which include coma, intracerebral hemorrhage and thrombosis of the deep venous system.\(^1\) Endovascular thrombolysis may be considered in patients with these risk factors, but its efficacy has not been proven in a randomized trial. Furthermore, in our experience endovascular thrombolysis is not beneficial to patients with impending transtentorial herniation due to large infarcts or hemorrhages\(^2\): It comes too late and cannot prevent further brainstem compression. Therefore, halfway 2006, we decided to change our policy and treat these patients with decompressive hemicraniectomy.\(^2\)

In this case series we describe the first 3 consecutive patients, discuss previous case reports and suggest a course of action for future research.

Methods

Since July 2006, we treated patients with severe CVT and signs of transtentorial herniation with decompressive hemicraniectomy. Indications for surgery are unilateral third nerve dysfunction and/or deterioration on the Glasgow coma score caused by local brain edema or venous infarction with midline shift or obliteration of basal cisterns, and not attributable to seizures.

A large hemicraniectomy was performed, with special effort to extend the decompression towards the temporal skull base. The dura was opened widely to ensure maximal decompression. The cortical surface was covered with haemostatic material (Surgicel\(^\text{®}\)), after which the skin, temporal muscle and fascia flap were closed in 3 layers. Patients received high dose, subcutaneous nadroparin immediately after the diagnosis CVT was made. Postoperatively, nadroparin was continued in prophylactic dosage for 24 hours. Thereafter, dosage was increased to therapeutic range. Patient C only received nadroparin after surgery, since she was operated immediately after admission.
Follow-up visits were performed at 6 and 12 months after discharge and outcome was expressed on the modified Rankin Scale (mRS, 0=complete recovery; 6=death).

Results

Patient A, a 39 year old man, was admitted with severe headache, nausea and disorientation (E4M6V5). His history included a deep-vein thrombosis of the left leg and a protein C deficiency. The CT-scan showed a left temporal hemorrhagic infarct (Figure 1A) and MR-V showed thrombosis of the left transverse and sigmoid sinuses. Despite nadroparin treatment, he deteriorated and became comatose (E1M3V1), due to enlargement of the hemorrhagic infarct with a 12 mm midline shift (Figure 1B). Hemicraniectomy was subsequently performed. Immediately post-operative the patient showed marked improvement (E3M5Vaphasia) and the CT-scan showed reduction of midline shift (figure 1C). At 6 months a right upper quadrant-anopia and a mild expressive aphasia were his only residual symptoms (mRS 2). At 12 months he had resumed all daily activities (mRS 1).

Patient B, a 36 year old woman, was admitted because of a generalized epileptic seizure (E4M6V5), after a week of headache and nausea. MR-V showed thrombosis of the superior sagittal sinus and a right-sided, parieto-occipital hemorrhagic infarct (volume 96 cm³). Despite nadroparin treatment, she deteriorated and on day 3 developed a depressed consciousness (E3M6V4) and an enlarging right pupil. CT-scan showed enlargement of the hemorrhagic infarct (133 cm³) and a midline shift of 9 millimeter. After emergent hemicraniectomy, the patient’s coma score optimized and the pupils became symmetrical. At 6 and 12 month follow-up a quadrant-anopia was her only residual symptom (mRS 1).

Patient C, a 55 year old woman, was found unconscious at home. It was estimated that she had been in coma for at least 12 hours. At examination she was comatose (E1M5V1), had a fixed and dilated left pupil and bilateral absent corneal reflexes. CT-scan showed a large left temporal hemorrhagic infarct (volume 134 cm³), with uncal herniation and a midline shift of 15 millimeter. The contrast enhanced CT-scan showed a thrombosis of the left transverse and
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Despite immediate hemicraniectomy, her clinical condition deteriorated in the following days (E,M, V). On day 3, treatment was withdrawn because there was no hope for recovery. She died 5 days later (mRS 6). The diagnosis of CVT was confirmed at autopsy.

Figure 1

A: Admission head CT-scan shows left temporal hemorrhagic infarct (34 cm³); B: CT-scan acquired after clinical deterioration, showing enlargement of hemorrhagic infarct (110 cm³) and increase of midline shift (12 mm); C: Direct post-operative CT scan. Reduction in midline shift (7 mm); D: Follow-up CT-scan 3 months after ictus

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Discussion

We present 3 consecutive cases with severe CVT and transtentorial herniation, treated with decompressive hemicraniectomy. This procedure resulted in excellent recovery in 2 patients. Before we changed our policy, similar patients in our center all had a fatal outcome despite maximal conservative treatment and endovascular thrombolysis.²

The scanty evidence for the efficacy of hemicraniectomy in CVT comes from small case series³⁻⁷, summarized in table 1. Including our cases, 11 out of 13 patients had a good outcome (mRS≤3). However, comparability between cases is hampered due to a wide variation in pre-operative clinical condition (GCS and pupillary reactions) among patients.

Table 1: Summary of case reports on decompressive hemicraniectomy in patients with severe CVT.

<table>
<thead>
<tr>
<th>Author</th>
<th>Year of publ.</th>
<th>No. cases</th>
<th>Age</th>
<th>GCS</th>
<th>Pupils</th>
<th>Favorable outcome (mRS ≤ 3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stefini³</td>
<td>1999</td>
<td>3</td>
<td>40-54</td>
<td>4-7</td>
<td>+/-</td>
<td>2/3</td>
</tr>
<tr>
<td>Barbati⁴</td>
<td>2003</td>
<td>1</td>
<td>15</td>
<td>5</td>
<td>+/-</td>
<td>1/1</td>
</tr>
<tr>
<td>Weber⁵</td>
<td>2004</td>
<td>1</td>
<td>62</td>
<td>NA</td>
<td>NA</td>
<td>1/1</td>
</tr>
<tr>
<td>Keller⁶</td>
<td>2005</td>
<td>4</td>
<td>37-66</td>
<td>6-13</td>
<td>+/-</td>
<td>4/4</td>
</tr>
<tr>
<td>Zeng⁷</td>
<td>2007</td>
<td>1</td>
<td>48</td>
<td>5</td>
<td>+/-</td>
<td>1/1</td>
</tr>
<tr>
<td>Current study</td>
<td>2008</td>
<td>3</td>
<td>36-55</td>
<td>5-13</td>
<td>variable</td>
<td>2/3</td>
</tr>
</tbody>
</table>

GCS = Glasgow Coma Score; mRS = modified Rankin Scale; NA = not available
* Bilateral hemicraniectomy performed in a patient with CVT without evident impending transtentorial herniation or mass lesions

There are several reasons why the concept of hemicraniectomy in severe CVT with impending herniation is plausible. First, hemicraniectomy can remove the immediate threat of fatal herniation. Second, decompressive hemicraniectomy has shown to be effective in young patients with malignant middle cerebral artery infarction and impending herniation.⁸ The mechanism causing death is likely to be similar in both diseases. Finally, there is ample evidence that even large venous infarcts in general have a better potential for recovery than arterial infarcts.

To obtain more reliable data, a prospective case registry of hemicraniectomy in CVT will be included in a new, large international study, the ISCVT-2.⁹
Participating centers will report clinical outcome on consecutive patients treated with decompressive hemicraniectomy for CVT. This will minimize selection bias of predominantly successful cases.

In conclusion, our data, supported by earlier case reports and pathophysiological plausibility, suggest that decompressive hemicraniectomy can be life-saving and result in an excellent outcome in the severest cases of CVT. Until more and better data are available, however, the decision to perform hemicraniectomy in CVT remains up to the individual judgment of the treating physician.

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References


