Should Decompressive Surgery Be Performed in Malignant Cerebral Venous Thrombosis?  
A Series of 12 Patients

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Background and Purpose—In malignant cerebral venous thrombosis (CVT) patients, emergency decompressive surgery has been suggested as a life-saving procedure. We report 12 patients with malignant CVT, among whom 8 underwent operation.

Methods—Retrospective study of 12 patients from 3 stroke units who had a malignant CVT as defined: (1) supratentorial cortical lesions attributable to superficial venous system thrombosis with or without sinus involvement; (2) with clinical (decreased consciousness and dilated pupils) or radiological signs of transtentorial herniation; (3) either at onset or after worsening despite heparin therapy. Surgery or abstention was decided individually by neurosurgeons on call.

Results—There were 9 women and 3 men with a mean age of 45.1 years. The delay between heparin therapy and signs of malignancy ranged from 2 to 30 hours. At malignant worsening all but 1 patient had hemorrhagic lesions; the median deviation of septum pellucidum was 12 mm (interquartile range, 6.7–13); 5 patients (including 3 who underwent operation) had a unilateral dilated pupil; and 4 (2 who underwent operation) had bilateral dilated pupils. Eight patients underwent surgical decompression, external decompression in 4, both external and internal decompression in 3, and internal decompression in 1. The 4 patients who did not undergo operation died within 1 to 5 days after diagnosis. One patient who underwent operation died of a pulmonary embolism. The 7 others survived, with, at last follow-up (median, 23.1 months; interquartile range, 19.7–45.6), an excellent recovery of mRS 0 or 1 in 6 and mRS 3 in 1.

Conclusion—Decompressive surgery may save lives and may even allow a good functional outcome in malignant CVT, even in patients with bilateral dilated pupils. (Stroke. 2010;41:00-00.)

Key Words: cerebral venous thrombosis  ■ decompressive surgery

Cerebral venous thrombosis (CVT) is a rare variety of cerebrovascular disease that can occur at any age and usually has a favorable outcome. However, some patients still have a poor outcome. In the prospective International Study on CVT cohort of 624 patients, death occurred in 8% and moderate to severe disability occurred in 5.1% of patients, despite the use of anticoagulant treatment in most patients. Transtentorial herniation is the most frequent cause of death. In patients with mass effect and midline shift who are worsening despite anticoagulation, emergency decompressive surgery has been suggested as a life-saving procedure. Because CVT is rare, and worsening despite treatment is extremely rare, a randomized controlled trial of hemicraniectomy, as successfully performed in malignant middle cerebral artery infarction, may not be feasible. We present a review of 12 patients with malignant CVT, among whom 8 underwent decompressive surgery.

Patients and Methods

Patients

From the 255 CVT patients seen between 2001 and 2008 in 3 stroke units in Paris, 12 patients (4.7%) with malignant CVT were prospectively collected in Lariboisière Hospital (8 of 176) and retrospectively in Bicêtre Hospital (3 of 33) and Tenon Hospital (1 of 16).

We defined malignant CVT as: supratentorial cortical lesions attributable to superficial venous system thrombosis with or without sinus involvement; with clinical (decreased consciousness and dilated pupils) or radiological signs of transtentorial herniation; either at onset or after worsening despite heparin therapy.
Twelve patients (9 females, 3 males), aged 18 to 68 years (mean 45 ± 15) satisfied the criteria for malignant CVT (Table 1; Supplementary Case Reports available online at http://stroke.ahajournals.org). A cause or risk factor was found in 11 patients. The main ones were oral contraception and hormonal replacement therapy. The first ever symptom was headache in 10 patients, seizure in 2, and focal deficit in 3. The time between first symptom and diagnosis ranged from 12 hours to 2 weeks (median, 5; IQR, 2.75–8). Other later symptoms included seizures in 5 patients, dysphasia in 5, right hemiplegia in 5, mutism in 2, and hemianopia in 1. Three patients (patients 1, 7, and 12) had signs of malignancy at onset or within 48 hours. The others had neurological symptoms (isolated headache in 7 patients, headache and focal deficit in 1, and headache, seizure, and fever in 1) for 3 to 14 days before a sudden or rapid worsening leading to investigations, CVT diagnosis, and heparin treatment. Signs of malignancy then occurred extremely rapidly in 2 to 30 hours (median, 5; IQR, 2.75–8). The diagnosis of CVT was based on conventional angiography, computed tomography venography, or MRI combined with MR venography, according to established diagnostic criteria.6

Decompressive Surgery
Surgery or abstention was decided individually by the neurosurgeons according to their current practice and availability. Surgical decompression was defined as an “external decompression” consisting of a large hemicraniectomy with durotomy, “internal decompression” if only resection of swollen or hemorrhagic brain tissue or hematoma evacuation was performed, or “external and internal decompression” if both types of surgery were performed. No comparative statistics were published, or “external and internal decompression” if both types of surgery were performed. No comparative statistics were published, or “external and internal decompression” if both types of surgery were performed. No comparative statistics were published, or “external and internal decompression” if both types of surgery were performed. No comparative statistics were published, or “external and internal decompression” if both types of surgery were performed. No comparative statistics were published, or “external and internal decompression” if both types of surgery were performed. No comparative statistics were published, or “external and internal decompression” if both types of surgery were performed.

Site of Thrombosis and Baseline Imaging Characteristics
By definition, all patients had cortical veins thrombosis. Superior sagittal sinus was involved in 6 cases, lateral sinus in 7 (right in 1, left in 5, and both in 1), and straight sinus in 1 case (Table 2). Nine patients had a parenchymal lesion at admission, which was hemorrhagic in 8 cases. Lesion was unilateral in 6 cases (frontal in 3, parietal in 2, parieto-temporal in 1). Table 1. Baseline Clinical Characteristics of Patients

<table>
<thead>
<tr>
<th>N</th>
<th>Gender</th>
<th>Age</th>
<th>Center</th>
<th>Risk Factors</th>
<th>Cause</th>
<th>First Symptom</th>
<th>GCS at Admission</th>
<th>Other Symptoms</th>
<th>Time to Diagnosis of Malignancy, d</th>
<th>Time of COVID to Malignancy, hr</th>
<th>GCS at Malignancy</th>
<th>Fixed Dilated Pupil at Malignancy</th>
<th>Time of Surgery, hr</th>
<th>GCS Before Surgery</th>
<th>6-Month Follow-Up mRS</th>
<th>Last Follow-Up mRS</th>
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<tr>
<td>1</td>
<td>F</td>
<td>68</td>
<td>Lrb</td>
<td>HRT, MTHFR</td>
<td>mutation</td>
<td>Seizure, confusion</td>
<td>14</td>
<td>Dysphasia, R hemiplegia</td>
<td>0.5</td>
<td>20</td>
<td>10</td>
<td>+</td>
<td>72</td>
<td>Sedation</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>22</td>
<td>Lrb</td>
<td>SLE, smoking</td>
<td></td>
<td>Seizure, fever, headache</td>
<td>14</td>
<td>7</td>
<td>24</td>
<td>5</td>
<td>+</td>
<td>60</td>
<td>Sedation</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>46</td>
<td>Lrb</td>
<td>Family history of DVT</td>
<td></td>
<td>Headache</td>
<td>9</td>
<td>Seizures, mutism, confusion</td>
<td>11</td>
<td>30</td>
<td>7</td>
<td>+</td>
<td>32</td>
<td>Sedation</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>18</td>
<td>KB</td>
<td>OC</td>
<td></td>
<td>Headache</td>
<td>10</td>
<td>Seizures, dysphasia, R hemiplegia</td>
<td>6</td>
<td>26</td>
<td>5</td>
<td>–</td>
<td>44</td>
<td>Sedation</td>
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<td>1</td>
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<tr>
<td>5</td>
<td>F</td>
<td>49</td>
<td>Lrb</td>
<td>OC</td>
<td></td>
<td>Headache, ICH</td>
<td>15</td>
<td>R hemiplegia, seizures</td>
<td>3</td>
<td>30</td>
<td>7</td>
<td>+/+</td>
<td>40</td>
<td>Sedation</td>
<td>6</td>
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</tr>
<tr>
<td>6</td>
<td>F</td>
<td>57</td>
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<td>HRT</td>
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<td>Headache, left hemiplegia</td>
<td>15</td>
<td>3</td>
<td>24</td>
<td>9</td>
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<tr>
<td>7</td>
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<td>Tnn</td>
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<td>Headache</td>
<td>13</td>
<td>Hemianopia, ICH</td>
<td>2</td>
<td>8</td>
<td>10</td>
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<td>24</td>
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<td>M</td>
<td>19</td>
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<td>APL syndrome</td>
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<td>Headache</td>
<td>14</td>
<td>Dysphasia, R hemiplegia</td>
<td>5</td>
<td>5</td>
<td>7</td>
<td>+/+</td>
<td>8</td>
<td>7</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>9</td>
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<td>32</td>
<td>Lrb</td>
<td>Pregnancy, spontaneous abortion</td>
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<td>Headache, ICH</td>
<td>14</td>
<td>Seizure</td>
<td>5</td>
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<td>6</td>
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<td></td>
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<tr>
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<td>46</td>
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<td>Headache</td>
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<td>Seizures</td>
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<td>24</td>
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<td>+</td>
<td></td>
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<td></td>
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<td>53</td>
<td>Lrb</td>
<td>Headache</td>
<td></td>
<td>Headache</td>
<td>15</td>
<td>Dysphasia</td>
<td>14</td>
<td>3</td>
<td>8</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>12</td>
<td>F</td>
<td>45</td>
<td>KB</td>
<td>OC, iron deficiency</td>
<td></td>
<td>Headache</td>
<td>7</td>
<td>Mutism</td>
<td>1.5</td>
<td>12</td>
<td>7</td>
<td>+/+</td>
<td></td>
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<td></td>
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</tr>
</tbody>
</table>

Median (IQR) 14 (12–15) 5 (2.7–8) 22 (7.2–24.5) 7 (6–9) 36 (28.5–48) 8 (7.5–9) 3 (1.5–3) 1 (0.5–1)

APL indicates antiphospholipid; DVT, deep venous thrombosis; F, female; GCS, Glasgow Coma Score; HRT, hormone replacement therapy; ICH, intracranial hypertension; IQR, interquartile range; KB, Kindbod Hospital; Lrb, Lariboisière Hospital; M, male; MTHFR, methylenetetrahydrofolate reductase; OC, oral contraception; R, right; SLE, systemic lupus erythematosus; Tnn, Ténon Hospital.

The diagnosis of CVT was based on conventional angiography, computed tomography venography, or MRI combined with MR venography, according to established diagnostic criteria.

The same neuroradiologist (G.S.) reviewed all neuroimaging data at admission and at the time of worsening. Parenchymal lesions were classified as hemorrhagic or nonhemorrhagic, depending on blood detection either on CT scan or T2*. MRI. Volume of parenchymal lesion (including edema and parenchymal hemorrhages) was calculated as described by Khotari et al6 for intracerebral hematoma.

Clinical outcome was measured according to the modified Rankin Score (mRS): complete recovery (mRS 0–1); partial recovery, independency (mRS, 2); dependency (mRS, 3–5), and death (mRS, 6). Functional outcome was evaluated at 6 months and at a last median follow-up of 23.1 months (interquartile range [IQR], 19.7–45.6).
poral in 1, and frontal bilateral in 3). The median volume of parenchymal lesions was 59 mL (IQR, 1.2–115.2). Seven patients had a subarachnoid hemorrhage, which was isolated in 2 patients. Eight patients had a midline shift with a median deviation of septum pellucidum of 2.7 mm (IQR, 0–6.7) (Figure).

**Imaging Data at Malignant Worsening**

All patients had parenchymal lesions, which were hemorrhagic in all but 1. Five had a new parenchymal lesion compared to baseline imaging. The volume of parenchymal lesions ranged from 57 mL to 262.2 mL (median, 144; IQR, 107.2–173). All patients had a midline shift with a median deviation of septum pellucidum of 12 mm (IQR, 6.7–13). Parenchymal lesion volume worsened by a mean of 9.1 mL per hour (range, 0.65–60.4) and severity of midline shift by 0.3 mm per hour (range, 0–0.8) (Figure).

**Decompressive Surgery**

Four patients did not undergo operation because the responsible neurosurgeons thought the situations were too severe for the patients to undergo surgery. The 8 others underwent operation. Four had an “external decompression,” 3 had both “external and internal decompression,” and 1 had only an “internal decompression.” Among the 4 latter, 3 had resection of brain tissue besides hematoma evacuation. Surgery was performed with a median delay of 36 hours (IQR, 28.5–48) after heparin initiation. Two patients who did not undergo operation and 3 who underwent operation had a unilateral fixed dilated pupil; 2 nonoperated and 2 operated patients had bilateral, fixed, dilated pupils at the time of malignant worsening.

**Comparison of Patients Who Did or Did Not Undergo Operation**

Demographic data were similar in the 2 groups (Table 3). Isolated headache was the first symptom in all 4 nonoperated patients, whereas 4 out of the 8 patients who underwent operation presented with more specific symptoms (including focal deficit, seizure, intracranial hypertension). This could explain a longer time to diagnosis (median, 9.5 days compared to 4) in the nonoperated group, which also had a shorter time to malignant worsening (median, 7.5 hours compared to 24). At baseline, the volume of parenchymal lesions was higher in the nonoperated group (median, 123 mL vs 43.5), as was the severity of midline shift (median, 6.5 mm vs 0.75). At worsening, the differences persisted between the 2 groups, with a median volume of lesions of, respectively, 164 mL vs 109 and a median midline shift of 12.5 mm vs 10. Progressions of midline shift and lesion volume were faster in the nonoperated group (median, 0.39 mm/hr vs 0.36 and 9.2 mL/hr vs 2.7).

**Follow-Up and Outcome**

The 4 nonoperated patients died within 1 to 5 days after diagnosis (median, 3.25; IQR, 2.5–4.25). All operated patients received intravenous heparin after the surgery at a median delay of 12 hours (IQR, 9–23). In the operated group, 1 patient (patient 5) who was neurologically improving died at day 9 of pulmonary embolism despite anticoagulation. The 7 others survived. They stayed in the intensive care unit from 4 to 52 days (median, 9.5 days; IQR, 5.7–29.2). The length of hospital stay (acute neurological care and rehabilitation) lasted from 27 days to 10 months (median, 148 days; IQR, 34.5–198.7). Five patients were discharged to a rehabilitation

### Table 2. Imaging Characteristics at Baseline and at Malignancy

<table>
<thead>
<tr>
<th>N</th>
<th>Site of Thrombosis</th>
<th>Topography</th>
<th>Type, H/NH</th>
<th>Volume, mL</th>
<th>Midline Shift, mm</th>
<th>Topography</th>
<th>Type, H/NH</th>
<th>Volume, mL</th>
<th>Midline Shift, mm</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>SSS, CV</td>
<td>Frontal</td>
<td>H</td>
<td>128</td>
<td>6</td>
<td>Frontal</td>
<td>H</td>
<td>182</td>
<td>15</td>
</tr>
<tr>
<td>2</td>
<td>DCV, CV</td>
<td>Frontal</td>
<td>NH</td>
<td>35</td>
<td>0</td>
<td>Frontal-parietal</td>
<td>NH</td>
<td>105.5</td>
<td>10</td>
</tr>
<tr>
<td>3</td>
<td>SSS, CV</td>
<td>Frontal</td>
<td>H</td>
<td>89</td>
<td>10</td>
<td>Frontal</td>
<td>H</td>
<td>109</td>
<td>13.1</td>
</tr>
<tr>
<td>4</td>
<td>SSS, RLS, CV</td>
<td>Parietal</td>
<td>H</td>
<td>66</td>
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<td>Parietal</td>
<td>H</td>
<td>110</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>LLS, CV</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>Temporoparietal</td>
<td>H</td>
<td>NA</td>
<td>Present</td>
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<tr>
<td>6</td>
<td>CV</td>
<td>Frontal bilateral</td>
<td>H</td>
<td>1.65</td>
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<td>Frontal bilateral</td>
<td>H</td>
<td>57</td>
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<td>Parieto-temporal</td>
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<td>52</td>
<td>6</td>
<td>Parieto-temporal</td>
<td>H</td>
<td>175.5</td>
<td>13</td>
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<tr>
<td>8</td>
<td>LLS, CV</td>
<td>0</td>
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<td>0</td>
<td>0</td>
<td>Temporoparietal</td>
<td>H</td>
<td>91</td>
<td>6</td>
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<tr>
<td>9</td>
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<td>Frontal bilateral</td>
<td>H</td>
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<td>H</td>
<td>144</td>
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<td>10</td>
<td>LLS, CV</td>
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<td>0</td>
<td>Fronto-parieto-temporal</td>
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<td>11</td>
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<td>Parietal</td>
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<td>135</td>
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<td>Parietal</td>
<td>H</td>
<td>170.6</td>
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<tr>
<td>12</td>
<td>SSS, CV</td>
<td>Frontal bilateral</td>
<td>H</td>
<td>111</td>
<td>9.5</td>
<td>Frontal bilateral</td>
<td>H</td>
<td>262.2</td>
<td>12</td>
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<tr>
<td>Median value (IQR)</td>
<td>59 (1.2–115.2)</td>
<td>27 (0–6.7)</td>
<td>144 (107.2–173)</td>
<td>12 (6.7–13)</td>
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</table>

CV indicates cortical veins; DCV, deep cerebral veins; H, hemorrhagic; LLS, left lateral sinus; NA, not available; NH, nonhemorrhagic; RLS, right lateral sinus; SSS, superior sagittal sinus. For patient 5, we only had written imaging data.
unit and the 2 others were discharged to home. At 6 months, the median mRS was 3 (2 patients with mRS 1; 1 patient with mRS 2; 3 patients with mRS 3; and 1 patient with mRS 5).

**Long-Term Follow-Up**

At last follow-up (median, 23.1 months; IQR, 19.7–45.6), the median mRS was 1, with 6 patients who had a total recovery (mRS, 0 or 1) and 1 (patient 4) who was still dependent (mRS, 3). Four out of the 7 surviving patients returned to a paid job (or study). Five patients had a reconstructive cranioplasty at a median delay of 9 months (IQR, 5.5–10). It consisted in a cementoplasty in 5 patients and an autograft in 1 (patient 2). However, the autograft had to be removed because of infection and afterward was replaced by a cementoplasty. Patient 8 had to undergo operation again after his first cementoplasty because of flap instability.

**Discussion**

Our study is the largest series so far to our knowledge to show that decompressive surgery (either external or internal or both) can be life-saving in malignant CVT and can allow a good functional recovery, thus confirming smaller case series or individual reports.\(^7\)–\(^{14}\) The usual overall prognosis of CVT is good, with a complete recovery in the majority of cases (79% in International Study on CVT\(^2\)). However, there are a number of severe cases either because of the site of thrombosis (cerebellar veins, deep venous system) or because of an underlying severe etiology or because of a rapid worsening leading to transtentorial herniation. In the present study, we used the term malignant CVT to designate a subset of severe CVT involving cortical veins, with or without sinus involvement, with supratentorial parenchymal lesions and signs of transtentorial herniation. Such cases are infrequent (4.5% in the Lariboisière prospective series).

Signs of malignancy may be present at onset or in the first 48 hours (25% of our cases) but usually occur after a few days of undiagnosed headache (60% of our cases). Once they

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**Figure.** Patient 4. 1 to 2, CT scan on day 0. Minimal mass effect without temporal herniation.\(^1\) Parietal hyperdensity (arrow) related to a hemorrhagic lesion attributable to superior sagittal sinus thrombosis.\(^2\) 3 to 4, CT scan day 1. Increased mass effect with temporal herniation (white arrow) and midline shift (small arrows). 5 to 7, CT scan on day 10 after decompressive craniectomy (*). No herniation. 8 to 9, Brain MRI at month 10 after cranioplasty: Parenchymatous sequelae (FLAIR\(^8\) and T2*-weighted\(^9\) hypointensities and hyperintensities).
appear, the deterioration is extremely rapid, with a median time between diagnosis (ie, heparin treatment) and signs of malignancy of 22 hours. Frequent seizures, the presence of large, mostly hemorrhagic parenchymal lesions, and a rapid increase in lesion volume seem suggestive of a malignant pattern of evolution.

In the absence of randomization, it is impossible to know what the outcome of the 4 nonoperated patients would have had if they had undergone operation, particularly because the cases were more severe at onset and at the time of worsening. Because of this imbalance and because the decision to operate was left to the treating neurosurgeon, any comparison between the 2 groups should be interpreted with caution. Nevertheless, it is remarkable that all 4 nonoperated patients died and that all but 1 (who died of pulmonary embolism) who underwent operation survived and improved neurologically, despite definite signs of temporal herniation and the presence of bilateral fixed dilated pupils in 2 cases. Initial improvement after surgery may occur within a few days but usually takes a few weeks. In contrast to arterial stroke patients, patients continue to improve very significantly months after surgery; 3 of our patients still had a mRS 3 at 6 months but several months later had a mRS 0 or 1. Overall, 6 of 7 patients had a mRS 0 or 1 at 1 year, which is a far better outcome than after hemicraniectomy for malignant middle cerebral artery infarction, although our patients underwent operation at a later stage.

Heparin is the treatment of choice in CVT; however, by definition, our cases deteriorated despite activated partial thromboplastin time (twice the controls) dose-adjusted intravenous heparin and, in 2 patients, despite thrombectomy and in situ fibrinolysis. The hypothesis may be raised that there is a collapse of cerebral veins attributable to malignant vaso-genic edema that restricts the effects of heparin and of other endovascular approaches.

There are several reasons why decompressive surgery is beneficial in malignant CVT. First, surgery immediately can remove the threat of fatal herniation. Second, because mass effect and elevated intracerebral pressure impair venous outflow and increase venous congestion, decompressive surgery may improve cortical collateral vein drainage, thus preventing the extension of thrombosis and possibly favoring the diffusion of heparin. Finally, there is a ample evidence from MRI diffusion studies that even large venous infarcts differ from arterial infarcts, with variable patterns of apparent diffusion coefficient maps explaining that venous infarcts in general have a far better potential for recovery than arterial infarcts. By saving lives, surgery allows the frequently favorable recovery observed in CVT.

In our series, the outcomes after external or internal decompression did not differ, but because of the small number of cases we cannot evaluate the benefits of each surgical approach. Whereas the removal of a large hematoma seems appropriate, resection of infarcted tissue is not justified given the aforementioned potential for good recovery of venous infarcts. In such cases, the less invasive technique of hemicraniectomy should be preferred.

In conclusion, CVT is an infrequent condition that, in ≈5% of cases, has a malignant course with signs of transtentorial herniation. In these malignant CVT, decompressive surgery may save lives and allow a good functional recovery. It should be performed as soon as possible when signs of malignancy appear, but survival with a good functional outcome is also possible in patients with bilateral dilated pupils. The presence of headache, and sometimes isolated headache, in the majority of cases for several days before the onset of signs of malignancy stresses the need for full neuroimaging investigations in patients with recent unusual persisting headache.

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Disclosure

None.

References

Supplemental Material

Case Reports

Patient 1 is a 68-year-old woman who had a sudden onset of partial seizures with altered consciousness attributable to a frontal hematoma. Angiography diagnosed superior sagittal sinus (SSS) and cortical veins thrombosis. Intravenous unfractionated heparin (UFH) was immediately started but a few hours later she had right hemiplegia and became comatose. SSS thrombosis was performed but the patient did not improve. Three days later, she had a left fixed dilated pupil and then underwent external and internal surgical decompression. She stayed 52 days in the intensive care unit and was discharged to a rehabilitation unit. She slowly improved (mRS 1 at 6 months) and recovered completely within 1 year. She was able to resume her medical practice (mRS, 0).

Patient 2 is a 22-year-old man with a 2-year history of thrombocytopenia. He first presented multiple seizures and brain MRI showed a right frontal edematous lesion. One week later, despite antiepileptic treatment, he had a generalized seizure. Brain MRI disclosed deep and cortical veins thrombosis, a new parietal lesion, and temporal herniation. Twenty-four hours later, he became comatose with a fixed dilated right pupil. He was intubated, anticoagulated, and underwent an external decompression. He stayed 4 days in the intensive care unit, rapidly improved, and was finally discharged home after 37 days. He had a mRS 1 at 6 months and finally had no sequelae (mRS 0 at 32 months).

Patient 3 is a 46-year-old woman who reported acute headaches. Eight days later, she had a generalized seizure, followed by aphasia. At admission, she presented multiple seizures, needing sedation and intubation. Brain MRI disclosed an edematous and hemorrhagic left frontal lesion with cortical veins and SSS thrombosis at MRA. She then received UFH. Thirty hours later, she had bradycardia and a left fixed dilated pupil. There was a transtentorial herniation on CT scan. She underwent an external decompression. The day after surgery, her condition deteriorated again with new frontal bleeding, needing an internal decompression. She stayed 51 days in the intensive care unit. She was discharged to a rehabilitation unit, where she is still hospitalized. She slowly improved but remained disabled with mRS 3 at 16 months.

Patient 4 is an 18-year-old woman who presented unusual persistent headaches. Six days later, she had generalized seizures followed by right hemiplegia and aphasia (Glasgow Coma Scale [GCS], 10). She was intubated. Brain CT scan disclosed a left parietal edematous and hemorrhagic lesion with SSS and right lateral sinus (LS) thrombosis. Despite UFH therapy, mannitol, and hypothermia, she deteriorated. CT scan disclosed temporal herniation. An external decompression was performed. She stayed 12 days in the intensive care unit and was able to go back home (mRS 3) after 6 months in rehabilitation. At 21 months, she had mRS 1 and had resumed her studies (Figure I).

Patient 5 is a 49-year-old woman with a 3-day history of headache. Brain CT scan disclosed a focal subarachnoid hemorrhage. Angiogram showed a left LS thrombosis. UFH was started but 2 hours later her condition deteriorated, with aphasia and partial seizures. The next day, she was comatose (GCS, 7), with a right hemiplegia and bilateral dilated pupil. Brain CT scan disclosed a voluminous left hemispheric edematous and hemorrhagic lesion with a temporal herniation. LS thrombectomy was unsuccessfully attempted and she underwent an external decompression. She started to improve neurologically but died 9 days later of pulmonary embolism.

Patient 6 is a 57-year-old woman who had a thunderclap headache followed by a partial left hemiplegia. Brain CT scan disclosed a localized subarachnoid hemorrhage. Angiography showed left cortical veins thrombosis. UFH was started. At day 2, her condition worsened, with a dense left hemiplegia and coma. Brain MRI disclosed bilateral frontal edematous and hemorrhagic lesions. She underwent an external decompression. She stayed 22 days in the intensive care unit and was able to go back home 8.5 months later after a long stay in a rehabilitation unit. Her mRS was 3 at 6 months and mRS was 1 at 2 years.

Patient 7 is a 55-year-old man who suddenly had dysphasia after 2 days of unusual headache. Brain MRI disclosed left LS thrombosis with a left temporoparietal hemorrhage. UFH was started. Eight hours later, he had a right hemiplegia and became comatose. Brain CT scan disclosed temporal herniation. He underwent an internal and external decompression. He stayed 5 days in ICU and was discharged home 27 days later. His mRS was 2 at 6 months and mRS was 1 at 5 years. However, he was not able to resume his previous high-responsibility job.

Patient 8 is a 19-year-old man who was treated by steroids and intravenous immunoglobulins for a severe antiphospholipid antibody syndrome, and by UFH for a concomitant pulmonary embolism. He had unusual headaches and 5 days later, he had a sudden aphasia with right hemiplegia. Brain CT scan disclosed a large edematous and hemorrhagic left temporal lesion. A few hours later he was comatose (GCS, 7), with bilateral fixed dilated pupils. He was sedated and intubated. Angiogram diagnosed left LS and cortical veins thrombosis. He underwent an external and internal decompression. He stayed 6 days in the intensive care unit and 10 months in a rehabilitation unit. He was discharged with mRS 3. He continued to slowly improve and had mRS 1 at 6.6 years.

Patient 9 is a 32-year-old woman who had had 2 spontaneous abortions and was pregnant (3 months) when she had unusual headaches and vomiting. Three days later, she had a generalized seizure. Brain MRI disclosed multiple frontal and parietal edematous and hemorrhagic lesions with a midline shift and despite SSS, right LS, and straight sinus thrombosis, the CTV diagnosis was not made initially. Final positive diagnosis was made 2 days later and, 2 hours after the beginning of UFH therapy, she was comatose, with bilateral fixed dilated pupils. Surgery was not performed because her condition was thought to be too severe. Despite UFH and mannitol treatments, she did not improve and she died from herniation 4 days later.

Patient 10 is a 46-year-old woman treated by intravenous chemotherapy for a lung carcinoma who presented unusual headaches and 2 weeks later had a partial seizure. Brain CT scan disclosed a diffuse subarachnoid hemorrhage with left LS thrombosis. She received full-dose low-molecular-weight heparin, but 24 hours later she had multiple seizures requiring sedation and intubation. CT scan disclosed a large left hemispheric hematoma with transtentorial herniation. During the next night, her left pupil dilated. She died 2 days later of herniation.

Patient 11 is a 53-year-old woman who had unusual persistent headaches and was pregnant (3 months) when she had unusual headaches and vomiting. Three days later, she had a generalized seizure. Brain MRI disclosed multiple frontal and parietal edematous and hemorrhagic lesions with a midline shift and despite SSS, right LS, and straight sinus thrombosis, the CTV diagnosis was not made initially. Final positive diagnosis was made 2 days later and, 2 hours after the beginning of UFH therapy, she was comatose, with bilateral fixed dilated pupils. Surgery was not performed because her condition was thought to be too severe. Despite UFH and mannitol treatments, she did not improve and she died from herniation 4 days later.

Patient 12 is a 44-year-old woman who reported unusual recent headaches. Twenty-four hours later, she was mutic, and a few hours later she was comatose (GCS, 7). Brain MRI disclosed bilateral frontal hemorrhagic infarcts with SSS thrombosis and transtentorial herniation. She received UFH. Twelve hours later, she had bilateral dilated pupils and sinusal tachycardia. Her condition was thought to be too severe for surgery. Despite intubation and mannitol therapy, she died within a few hours.
Should Decompressive Surgery Be Performed in Malignant Cerebral Venous Thrombosis? A Series of 12 Patients
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