Blood blister–like aneurysms of the internal carotid artery trunk causing subarachnoid hemorrhage: treatment and outcome

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Object. The object of this study was to evaluate cases of subarachnoid hemorrhage (SAH) from ICA aneurysms. A total of 211 patients suffered SAH from ICA aneurysms. Of these, 14 patients (6.6%) had ICA trunk aneurysms or ICA trunk aneurysm, or ICA trunk aneurysms. They are associated with preponderance of the right ICA, hypertension, arteriosclerosis, younger age, female dominance, and thin walls on pathologic examination. They are characterized by a half-dome or blood blister-like shape on angiography and thin walls on pathologic examination. Treatment of BBAs is associated with a high morbidity rate. As pointed out by Charbel et al., this is due to difficulty in making the diagnosis because of the small size of these aneurysms, a very high risk of intraoperative aneurysm rupture, and most importantly, a high probability of losing parent vessel patency, either intentionally or as a consequence of the treatment. The aim of this single-center, retrospective study was to evaluate the clinical and imaging characteristics as well as

Abbreviations used in this paper: ACA = anterior cerebral artery; ACaO = anterior communicating artery; BBA = blood blister–like aneurysm; CSF = cerebrospinal fluid; CT = computed tomography; DIND = delayed ischemic neurological deficit; DS = digital subtraction; EC–IC = extracranial–intracranial; GOS = Glasgow Outcome Scale; ICA = internal carotid artery; ICP = intracranial pressure; MCA = middle cerebral artery; PCoA = posterior communicating artery; SAH = subarachnoid hemorrhage; TBA = transluminal balloon angioplasty; TCD = transcranial Doppler ultrasound.
treatment outcomes of patients with acute SAH from BBA ICA trunk aneurysms. With respect to treatment outcomes, we focus in particular on outcomes in cases in which patients could be treated with preservation of their ICA versus cases in which the ICA was sacrificed.

**Clinical Materials and Methods**

**Patient Population**

A total of 921 subjects with acutely ruptured intracranial aneurysms were admitted to the Rikshospitalet (the national hospital of Norway) in Oslo between January 1998 and August 2005. Of these, 211 patients (22.9%) had ruptured ICA aneurysms. From this population, we identified 14 patients with aneurysms at nonbranching sites of the suprACLinoidal portion of the ICA (Fig. 1). These 14 patients, constituting 1.5 and 6.6% of all patients with ruptured aneurysms and ruptured ICA aneurysms, respectively, comprised our study population.

**Clinical Evaluation**

All patients were transferred to the Rikshospitalet from referring hospitals. The median time from start of SAH symptoms to arrival at our institution was 12 hours (range 3.8–93.0 hours). The patients were examined clinically on their arrival at our institution and again prior to aneurysm repair. Two individuals experienced repeated bleeding after SAH had been diagnosed but before undergoing aneurysm treatment, one before and one after arrival at our hospital. Their preoperative clinical condition was scored according to the Hunt and Hess grading scale. For those who arrived intubated, clinical condition just prior to intubation was recorded. In 6 of 14 patients, the preoperative Hunt and Hess grade was I or II (Table 1).

**Imaging Studies**

The patients were investigated radiologically at our institution by means of cerebral CT and cerebral CT angiography shortly after arrival, and all but one also underwent DS angiography.

Based on the preoperative CT findings, the patients’ condition was scored according to the Fisher scale as well as the modified Fisher scale. The cerebral CT findings were as follows (Table 2): Three individuals had SAH < 1 mm (Fisher Grade 2), 3 had SAH > 1 mm (Fisher Grade 3), while 8 had intracerebral hemorrhage and/or intraventricular hemorrhage (Fisher Grade 4). Using the modified Fisher scale, 6 patients had massive SAH as well as intracerebral hemorrhage and/or intraventricular hemorrhage (Fisher Grade 3 + 4).

Small ventricles were present in 8 patients (Evans ratio ≤ 0.30) and large ventricles in 6 (Evans ratio > 0.30). Data pertaining to the type, location, and size of the ICA aneurysms in these 14 patients are presented in Table 2. Thirteen aneurysms were classified by the neuroradiologists as broad-based or atypical blebs (BBAs) and 1 as a fusiform ectasia. The BBAs pointed medially or anteriorly in 11 patients (79%). In 10 patients (71%), the aneurysm was located on the right ICA. All BBAs were small (< 6 mm long). Only one patient had local signs of ICA dissection, whereas mild to moderate vasoaspsasm was demonstrated angiographically in 3 patients.

Preoperative evaluation of the cerebral circulatory collateral capacity was performed by means of DS angiography in 13 patients (Table 3); in 1 patient the evaluation was based only on the results of CT angiography. In all but 1 patient, the ACoA was > 1 mm. Eleven of the 13 patients evaluated had spontaneous cross-flow to the contralateral MCA as well as double filling of the ACAs on digital compression series and very little cross-flow to the contralateral MCA. In 3 patients the ipsilateral PCoA was not visualized, and in 1 patient it was only visible on the digital compression series. Another 4 patients had slim ipsilateral PCoAs.

With respect to tolerance to acute ICA closure, 10 of the 11 patients with good-caliber ACoAs and spontaneous cross-flow to the contralateral MCA (Fig. 1) were considered eligible (Table 3). One patient with a good-caliber ACoA had a delayed venous filling (that is, > 3 seconds) and was therefore considered ineligible. Two patients were considered ineligible for ICA closure due to poor ACoAs, whereas 1 patient did not undergo DS angiographic evaluation (Table 3). The CT angiography findings in this patient suggested that ICA sacrifice probably could be tolerated because of a good-caliber ACoA, although the ipsilateral PCoA could not be identified.

A preoperative hemodynamic evaluation of the cerebral circulatory collateral capacity was also performed in 4 subjects (Table 3) by means of TCD. Three of these 4 were considered able to tolerate ICA sacrifice, and 3 patients were noted to have moderate vasoaspsasms. We tried to keep ICP < 20 mm Hg in our patients. The algorithm used to treat raised ICP included sedation, drainage of CSF through the ventricular route, and hyperosmolar solutions (Hyper-Haes). In situations of uncontrollable ICP, a decompressive craniectomy could be performed.

We tried to ensure adequate cerebral perfusion pressure (> 70 mm Hg) in those patients in whom the ICA could be preserved and > 90 mm Hg in patients in whom the ICA was sacrificed through hyperdynamic cerebral circulatory treatment. The patients received intravenous nimodipine (Nimotop) the first 3 weeks after SAH. Patients treated with surgical clip placement received low–molecular weight heparin from the day after vessel closure (2500 IU Fragmin subcutaneously daily), whereas those treated endovascularly received an initial dose of 3000–5000 IU heparin and thereafter 1000 IU/hour on the day of treatment, 7500 IU Fragmin twice daily the following 2 days, 5000 IU twice daily the next 2 days, and thereafter 2500 IU Fragmin daily.

The patients who survived were transferred to their local neurological departments at the end of their treatment in our institution. They underwent routine clinical and neuro- radiological follow-up examinations at our department 3 and 12 months postoperatively. Outcome was scored according to the GOS.

**Statistical Analysis**

The Student t-test was used to compare numerical parameters for the 2 types of treatment (ICA preservation or ICA trapping). The Fisher exact probability test was used to compare proportional factors (sex, side, hypertension, ICA dissection, arteriosclerosis) for the 2 groups.
Results

Aneurysm Treatment

In 13 patients, endovascular treatment was attempted initially, the aneurysm in 1 patient was thought unsuitable for coil placement. Coil placement with preservation of the ICA was only successful in 2 (15%) of 13 patients. In 1 patient, coil placement failed and a coil ligation of the ICA was undertaken within 48 hours after the SAH (Table 4).

Thus, surgery was undertaken in 11 patients, with the intention of aneurysm clipping and parent vessel preservation. This could be achieved in 5 patients, 2 of whom were transported to Helsinki for treatment by one of the authors (J.H.). The ICA was trap-ligated in 6 patients because of profuse intraoperative bleeding from the BBA that emerged as a pathological segment of the ICA with a large visible defect/tear in the vessel wall. In all cases, the aneurysm clips were positioned across the ICA so as to preserve the PCoA. The trap ligation was performed within 48 hours after SAH (Table 4).

Thus, 7 patients were treated with preservation of ICA and 7 patients with ICA trapping. These 2 treatment groups were not statistically different with respect to patient age (p = 0.44), sex (p = 1.00), Glasgow Coma Scale score (p = 0.28), Hunt and Hess grade (p = 0.08), hypertension (p = 0.40), Fisher grade (p = 0.16), Evans ratio (p = 0.44), time from SAH to hospitalization at the Rikshospitalet (p = 0.35), aneurysm size (p = 0.54), ICA dissection (p = 0.50), arteriosclerosis (p = 0.50), collateral circulation (p = 1.00), time from arrival at Rikshospitalet to definitive aneurysm treatment (p = 0.24), time from SAH to treatment (p = 0.19), or mode of treatment (p = 0.40). However, a significant difference was noted with respect to the side of aneurysm location (p = 0.04).

Clinical Course After ICA Preservation

The 7 patients in whom the ICA could be preserved had a rather benign clinical course. Five patients were extubated shortly after treatment and made an unremarkable clinical recovery after SAH. One patient was maintained

### TABLE 1
Characteristics of 14 patients with BBAs and SAH*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Initial GCS Score†</th>
<th>Time from SAH to Arrival at RH (hrs)</th>
<th>Preop H &amp; H Grade</th>
<th>Comorbidities</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>29.9, F</td>
<td>4</td>
<td>3.8</td>
<td>V</td>
<td>juvenile RA</td>
</tr>
<tr>
<td>2</td>
<td>50.9, M</td>
<td>4</td>
<td>87.7</td>
<td>V</td>
<td>none</td>
</tr>
<tr>
<td>3</td>
<td>62.4, F</td>
<td>15</td>
<td>46.0</td>
<td>II</td>
<td>hypertension, mild bronchial asthma</td>
</tr>
<tr>
<td>4</td>
<td>47.5, M</td>
<td>3</td>
<td>7.0</td>
<td>V</td>
<td>none</td>
</tr>
<tr>
<td>5</td>
<td>67.7, F</td>
<td>14</td>
<td>18.5</td>
<td>II</td>
<td>bronchial asthma, migraine</td>
</tr>
<tr>
<td>6</td>
<td>42.1, F</td>
<td>14</td>
<td>32.5</td>
<td>V</td>
<td>none</td>
</tr>
<tr>
<td>7</td>
<td>52.8, M</td>
<td>6</td>
<td>5.0</td>
<td>V</td>
<td>none</td>
</tr>
<tr>
<td>8</td>
<td>48.4, F</td>
<td>4</td>
<td>4.3</td>
<td>V</td>
<td>none</td>
</tr>
<tr>
<td>9</td>
<td>47.8, F</td>
<td>14</td>
<td>22.7</td>
<td>IV</td>
<td>migraine</td>
</tr>
<tr>
<td>10</td>
<td>43.2, F</td>
<td>14</td>
<td>14.5</td>
<td>II</td>
<td>kidney transplant, hypertension, uterine cancer</td>
</tr>
<tr>
<td>11</td>
<td>55.8, F</td>
<td>10</td>
<td>22.0</td>
<td>III</td>
<td>mild bronchial asthma</td>
</tr>
<tr>
<td>12</td>
<td>47.8, M</td>
<td>15</td>
<td>14.0</td>
<td>II</td>
<td>none</td>
</tr>
<tr>
<td>13</td>
<td>42.8, M</td>
<td>15</td>
<td>5.0</td>
<td>I</td>
<td>none</td>
</tr>
<tr>
<td>14</td>
<td>37.7, M</td>
<td>15</td>
<td>93.0</td>
<td>II</td>
<td>hypertension, migraine</td>
</tr>
</tbody>
</table>

* GCS = Glasgow Coma Scale; H & H = Hunt and Hess; RH = Rikshospitalet, Oslo; RA = rheumatoid arthritis.
† Score at initial admission to the referring hospital.
on sedation and artificial ventilation for 11 days postoperatively due to respiratory problems, but subsequently made an unremarkable clinical recovery. The patient in Case 11 developed significant vasospasm postoperatively. She also had meningitis from her external ventricular drain and was left with a significant left hemiparesis. She was alert and oriented upon discharge to her local hospital after 3 weeks, but subsequently required shunt placement due to respiratory problems, but subsequently made a good recovery. A CT scan obtained on Day 1 showed no signs of cerebral ischemia, but vasospasm began to develop, as evidenced by TCD. The vasospasm increased in severity and the ICP increased steadily, despite pentothal sedation to burst suppression, maximal drainage of CSF through the ventricular route, active cooling, and treatment with hyperosmolar solutions. A CT scan obtained on Day 8 showed low attenuation in the ipsilateral MCA territory, and on the following day a massive infarct and brain shift were evident (Fig. 2). On Day 9, the patient underwent a hemicraniectomy, which resulted in a good outcome. There were no deaths in this group.

Clinical Course After ICA Sacrifice

The 7 cases involving acute-stage ICA sacrifice can be divided into 3 groups based on clinical course: The first group consists of the 2 patients (Cases 3 and 5) deemed to not tolerate ICA sacrifice on the preoperative DSA. Both patients experienced raised ICP and ischemic strokes directly after surgery, as verified by CT scans on Day 1, and died in the immediate postoperative period (Fig. 2). The patient in Case 5 died on Day 1 and the patient in Case 3 on Day 5. (For purposes of this article, days are numbered from the day of aneurysm treatment at Rikshospitalet.)

The second group consists of 4 cases in which ICA sacrifice was considered likely to be tolerated, based on preoperative DS angiography findings of good cross-filling of the MCA and ACA, good-sized ACoAs, and good-sized PCoAs. The first patient (Case 2) had a CT scan on Day 1 (the 1st day of hospitalization at Rikshospitalet, the 5th day after SAH) that showed no signs of cerebral ischemia, but vasospasm and raised ICP developed on Day 3, and an ipsilateral infarct was evident on the CT scan obtained that same day (Day 3). He underwent hemicraniectomy and survived with a poor outcome (GOS 3). The patient in Case 4 had good filling of the ipsilateral MCA as demonstrated by TCD on Day 1. A CT scan obtained on Day 1 demonstrated no low-attenuating areas (only very slight edema throughout the hemisphere), whereas a CT scan obtained on Day 2 showed low attenuation in the ipsilateral internal capsule area. On Day 5, he developed raised ICP and a CT scan obtained on Day 5 showed cerebral infarction in the ipsilateral MCA territory and a significant brain shift. He died on Day 6. In Case 6 vasospasm and raised ICP developed on Day 6 and ipsilateral infarct was evident on CT scans on Day 9. The patient underwent hemicraniectomy, but died on Day 11. The last patient in this group also had a very stormy clinical course (Case 7). On Day 3, a CT scan showed no low-attenuating foci (Fig. 3), but vasospasm began to develop, as evidenced by TCD. The vasospasm increased in severity and the ICP increased steadily, despite pentothal sedation to burst suppression, maximal drainage of CSF through the ventricular route, active cooling, and treatment with hyperosmolar solutions. A CT scan obtained on Day 8 showed low attenuation in the ipsilateral MCA territory, and on the following day a massive infarct and brain shift were evident (Fig. 2). On Day 9, the patient underwent a hemicraniectomy, which resulted in a temporary resolution of the raised ICP. He died on Day 20.

The third group includes only the single patient (Case 1) who did not undergo DS angiography and whose collateral circulation was evaluated only by means of CT angiography. A CT scan obtained on Day 2 showed no signs of cerebral ischemia. Nevertheless, the patient developed raised ICP early in the postoperative period, and ipsilateral cerebral infarcts were evident on CT on Day 3. She died on Day 4.

Outcome of Treatment

Only one patient among those in whom the ICA was preserved had a poor outcome. In contrast, outcome was very poor for the 7 patients treated with ICA closure, with 6 of them dying (GOS 1) and 1 surviving in poor condition (GOS 3). This difference was highly significant (p = 0.001, Fig. 4).

Discussion

In this single-center, retrospective study, 14 cases of blood blister–like ICA aneurysms were evaluated. The study confirms the low incidence of BBAs and the predominance of localization of the right ICA. Eight (57%) of the 14 patients were women, which contrasts somewhat the striking female preponderance (71–100%) found in stud-
ies from Asia. Although the Asian case series were small, thus making the significance of statistical differences uncertain, there could possibly be genetic differences between the Asian and the Norwegian patient cohorts that might explain the difference in the findings.

The aneurysms were < 6 mm in all patients, with an average size of 2.5 × 3.9 mm. The small sizes of these aneurysms, together with their configuration, led to difficulties in treating them endovascularly. Coil placement failed in 10 cases and was judged to be unsuitable in 1. Hence, 11 patients (79%) underwent surgery. These results contrast with those for the other ICA aneurysms treated in our institution, in which 57% of aneurysms were treated endovascularly in the same study period (unpublished results). Our experience, however, is in accordance with a recent report by Park et al., in which the authors concluded that endovascular coiling of BBAs of the ICA cannot be recommended due to the high rates of procedural rupture (75%), aneurysm regrowth, and rebleeding.

The most frightening characteristic of the BBAs was the high rate of intraoperative aneurysm rupture. Of the 11 patients treated surgically, aneurysm rupture during dissection occurred in 5 patients (45%). This is in accordance with findings of several other authors who have reported that clip placement surgery alone frequently provokes BBA avulsion and ICA laceration. Nevertheless, it contrasts with the 10% intraoperative aneurysm rupture rate during dissection of all other ruptured ICA aneurysms treated surgically in our institution in the same study period (unpublished results) and generally reported intraoperative aneurysm rupture rates of about 7%. Thus, the BBAs represent an especially malignant variant of aneurysm, carrying a high risk of rupture during surgery.

The aneurysm ruptures were diffuse tears in the vessel wall and invariably led to ICA trap ligation. Although attempts to preserve the parent artery were made, including wrapping in some instances and suturing in others, the thin vessel walls surrounding the tears made these efforts unsuccessful. The ICA was sacrificed as a last resort. As demonstrated by Sim et al., a high degree of preoperative awareness and extremely careful dissection during surgery can prevent poor clinical outcomes. Although they also experienced intraoperative rupture in 5 of 10 cases, they successfully placed clips to preserve the ICA in 2 cases, wrapped BBAs to preserve the ICA in 2 cases, and trap ligated the ICA in 1 case. Obviously, the ideal treatment of an ICA aneurysm is aneurysm repair with parent artery preservation. In our case series, this was achieved in 202 (96%) of 211 patients who suffered ICA aneurysm hemorrhage in the study period. Thus, the ICA was sacrificed in 9 cases, 7 of which are reported here.

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Evaluation</th>
<th>ACoA</th>
<th>Ipsilateral PCoA</th>
<th>Angio</th>
<th>TCD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>not performed</td>
<td>good caliber</td>
<td>not visible</td>
<td>probably no</td>
<td>not evaluated</td>
</tr>
<tr>
<td>2</td>
<td>DSA w/ dig compr &amp; TCD</td>
<td>good caliber; double filling of distal ACAs &amp; MCA</td>
<td>slim</td>
<td>yes</td>
<td>not evaluated</td>
</tr>
<tr>
<td>3</td>
<td>DSA w/ dig compr</td>
<td>very good caliber; double filling of distal ACAs &amp; MCA</td>
<td>not visible</td>
<td>slow venous phase (&gt;3 sec)</td>
<td>not evaluated</td>
</tr>
<tr>
<td>4</td>
<td>DSA w/ dig compr &amp; TCD</td>
<td>very good caliber; double filling of distal ACAs &amp; MCA</td>
<td>good caliber</td>
<td>yes</td>
<td>not evaluated</td>
</tr>
<tr>
<td>5</td>
<td>DSA w/ dig compr</td>
<td>not visible</td>
<td>slim</td>
<td>no</td>
<td>not evaluated</td>
</tr>
<tr>
<td>6</td>
<td>DSA w/ dig compr</td>
<td>very good caliber; double filling of distal ACAs &amp; MCA</td>
<td>very slim</td>
<td>yes</td>
<td>not evaluated</td>
</tr>
<tr>
<td>7</td>
<td>DSA w/ dig compr</td>
<td>very good caliber; double filling of distal ACAs &amp; MCA</td>
<td>good caliber</td>
<td>yes, rapid venous phase</td>
<td>not evaluated</td>
</tr>
<tr>
<td>8</td>
<td>DSA w/ dig compr &amp; TCD</td>
<td>good caliber; double filling of distal ACAs &amp; MCA</td>
<td>not visible</td>
<td>yes</td>
<td>not evaluated</td>
</tr>
<tr>
<td>9</td>
<td>DSA w/ dig compr</td>
<td>very good caliber; double filling of distal ACAs &amp; MCA</td>
<td>slim</td>
<td>yes</td>
<td>not evaluated</td>
</tr>
<tr>
<td>10</td>
<td>DSA w/ dig compr</td>
<td>good caliber; double filling of ACAs &amp; some filling of contralateral MCA</td>
<td>good caliber</td>
<td>yes, rapid venous phase</td>
<td>not evaluated</td>
</tr>
<tr>
<td>11</td>
<td>DSA w/ dig compr</td>
<td>very good caliber; double filling of distal ACAs &amp; MCA</td>
<td>good caliber</td>
<td>yes</td>
<td>not evaluated</td>
</tr>
<tr>
<td>12</td>
<td>DSA w/ dig compr</td>
<td>good caliber; double filling of distal ACAs &amp; MCA</td>
<td>good caliber</td>
<td>yes</td>
<td>not evaluated</td>
</tr>
<tr>
<td>13</td>
<td>DSA w/ dig compr</td>
<td>very good caliber; double filling of distal ACAs &amp; MCA</td>
<td>good caliber</td>
<td>yes, very good collaterals</td>
<td>not evaluated</td>
</tr>
<tr>
<td>14</td>
<td>DSA w/ dig compr &amp; TCD</td>
<td>slim, but double filling of distal ACAs on dig compr series</td>
<td>visible only on dig compr series</td>
<td>no</td>
<td>no; moderate vasospasm</td>
</tr>
</tbody>
</table>

* angio = angiography; contralat = contralateral; DSA = digital subtraction angiography; dig compr = digital compression.
† Assessed by means of conventional cerebral angiography and TCD.

Table 3: Results of preoperative evaluation of cerebral collateral vessels.
## Table 4

**Summary of aneurysm treatment and outcome**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Time from SAH to Rx (days)</th>
<th>Time from Arrival to Rx (hrs)</th>
<th>Acute ICA Closure</th>
<th>Aneurysm Treatment</th>
<th>Postop CT Findings</th>
<th>TCD Findings</th>
<th>Clinical Course</th>
<th>GOS Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.3</td>
<td>2.3</td>
<td>yes</td>
<td>ICA trap ligation (intraop rupture)</td>
<td>Day 2: no low-atten foci; Day 3: low atten in ipsilat MCA &amp; ACA terr</td>
<td>NA</td>
<td>RICP fr Day 1 on; died Day 4</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>4.7</td>
<td>25.7</td>
<td>yes</td>
<td>ICA trap ligation (intraop rupture) after failed coil pl attempt</td>
<td>Day 2: no low-atten foci; Day 3: low atten in ipsilat MCA terr</td>
<td>vasospasm fr Day 5 to Day 9</td>
<td>hemianctectomy Day 3 due to RICP</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>2.4</td>
<td>11.0</td>
<td>yes</td>
<td>ICA trap ligation</td>
<td>Day 2: low atten in ipsi- &amp; contralat hems</td>
<td>NA</td>
<td>died Day 5</td>
<td>1</td>
</tr>
<tr>
<td>4</td>
<td>1.8</td>
<td>36.8</td>
<td>yes</td>
<td>ICA trap ligation (intraop rupture) after failed coil pl attempt</td>
<td>Day 1: edema in ipsilat hem; Day 2: low-atten focus in ipsilat int cap; Day 5: low atten in ipsilat MCA terr &amp; significant brain shift</td>
<td>good flow in ipsilat MCA Day 1</td>
<td>severe RICP fr Day 3; bilat dilation of pupils Day 5; died Day 6</td>
<td>1</td>
</tr>
<tr>
<td>5</td>
<td>1.4</td>
<td>14.1</td>
<td>yes</td>
<td>ICA trap ligation (intraop rupture)</td>
<td>Day 1: low atten in ipsilat MCA terr, massive brain shift</td>
<td>not performed</td>
<td>died Day 1</td>
<td>1</td>
</tr>
<tr>
<td>6</td>
<td>1.7</td>
<td>7.6</td>
<td>yes</td>
<td>ICA trap ligation (intraop rupture)</td>
<td>Day 2: no low-atten foci &amp; good flow in ipsilat MCA &amp; ACA terrs; Day 5: no low-atten foci; Day 9: low atten in ipsilat ACA &amp; MCA terrs; Day 11: low atten in ipsi- &amp; contralat hems</td>
<td>vasospasm fr Day 6</td>
<td>severe RICP fr Day 6; bilat dilation of pupils Day 8; hemianctectomy Day 9; died Day 11</td>
<td>1</td>
</tr>
<tr>
<td>7</td>
<td>0.4</td>
<td>5.0</td>
<td>yes</td>
<td>ICA coil ligation</td>
<td>Day 3: no low-atten foci; Day 8: low atten in ipsilat MCA terr; Day 9: low atten in ipsilat MCA terr, massive brain shift</td>
<td>vasospasm fr Day 3</td>
<td>hemianctectomy Day 9; died Day 20</td>
<td>1</td>
</tr>
<tr>
<td>8</td>
<td>1.1</td>
<td>22.8</td>
<td>no clip</td>
<td>ICA trap ligation (intraop rupture)</td>
<td>Day 2: no low-atten foci; Day 4: no low-atten foci; Day 7: no low-atten foci; Day 10: no low-atten foci; Day 23: no low-atten foci</td>
<td>not performed</td>
<td>extubated &amp; in good neuro cond on Day 2; reintubated later same day due to respiratory distress; off ventilator Day 11, thereafter unremarkable clinical recovery</td>
<td>5</td>
</tr>
<tr>
<td>9</td>
<td>1.4</td>
<td>10.3</td>
<td>no coil</td>
<td>ICA trap ligation (intraop rupture)</td>
<td>Day 1: no low-atten foci; Day 5: no low-atten foci; Day 11: no low-atten foci</td>
<td>not performed</td>
<td>extubated &amp; in good neuro cond on Day 1; acute hemiparesis Day 8; quick spontaneous improvement, thereafter unremarkable clinical recovery</td>
<td>5</td>
</tr>
<tr>
<td>10</td>
<td>0.9</td>
<td>6.5</td>
<td>no coil</td>
<td>ICA trap ligation (intraop rupture)</td>
<td>Day 8: no low-atten foci, vasospasm on CTA; Day 16: no low-atten foci, no vasospasm</td>
<td>not performed</td>
<td>extubated &amp; in good neuro cond on Day 1; acute hemiparesis Day 8; quick spontaneous improvement, thereafter unremarkable clinical recovery</td>
<td>5</td>
</tr>
<tr>
<td>11</td>
<td>3.4</td>
<td>59.0</td>
<td>clip after transfer to Helsinki</td>
<td>ICA trap ligation (intraop rupture)</td>
<td>Day 2: no low-atten foci, but vasospasm on DSA; Day 12: low-atten focus in ipsilat int cap</td>
<td>not performed</td>
<td>postop vasospasm, meningitis, 2</td>
<td>2</td>
</tr>
<tr>
<td>12</td>
<td>17.3</td>
<td>401.0</td>
<td>clip after initial angiography</td>
<td>no clip after initial angiography was negative, but ndt angiography demonstrated aneurysm</td>
<td>Day 1: no low-atten foci</td>
<td>not performed</td>
<td>extubated &amp; in good neuro cond on Day 1; unremarkable, good clinical recovery</td>
<td>5</td>
</tr>
<tr>
<td>13</td>
<td>1.0</td>
<td>18.0</td>
<td>no clip</td>
<td>ICA trap ligation (intraop rupture)</td>
<td>Day 3: no low-atten foci; Day 5: no low-atten foci; Day 7: no low-atten foci; Day 11: no low-atten foci</td>
<td>not performed</td>
<td>extubated &amp; in good neuro cond on Day 1</td>
<td>5</td>
</tr>
<tr>
<td>14</td>
<td>7.4</td>
<td>84.0</td>
<td>clip after transfer to Helsinki</td>
<td>ICA trap ligation (intraop rupture)</td>
<td>Day 1: no low-atten foci</td>
<td>preop TCD demonstrated intolerance to ICA sacrifice</td>
<td>extubated &amp; in good neuro cond on Day 1; unremarkable, good clinical recovery</td>
<td>5</td>
</tr>
</tbody>
</table>

* Day numbers refer to days after treatment, not SAH. Abbreviations: atten = attenuating or attenuation; cond = condition; CTA = CT angiography; fr = from; hem = hemisphere; hydroceph = hydrocephalus; ICU = intensive care unit; int cap = internal capsule; intraop = intraoperative; NA = not available; neurol = neurological; pl = placement; rpd = repeated; Rx = therapy; RICP = raised ICP; terr = territory.
terrible, and most often lethal, second insult to the brain. In some patients, poor outcome was related to unfavorable vascular anatomy, with ICA sacrifice causing an immediate ischemic stroke. In others, in whom DS angiography with anatomical evaluation of ACoAs and PCoAs demonstrated favorable cerebral collateral circulation, massive late-onset cerebral infarctions in the circulatory territory of interest developed. The infarcts and subsequent raised ICP were the final causes of death, but this chain of events occurred only some days after vessel closure and concurred with the time frame of cerebral vasospasm.38,53 Serial TCD measurements were performed in the majority of these cases, demonstrating vasospasm. Although action was taken to avoid decreases in cerebral blood flow, the vasospasm occurred on top of a susceptible hemodynamic situation caused by the closure of a major precerebral vessel. Thus, the lethal second insult was probably due to the vasospasm’s strangulation of the cerebral collateral blood vessels. In addition, MCA vasospasm will further decrease an already lowered regional cerebral blood flow in the MCA perfusion territory. Consequently, we hypothesize that vasospasm may be the final cause of cerebral infarction and hence death in these patients.

Even in the presence of preoperative angiographic test results suggesting adequate collateral capacity, ICA sacrifice cannot be safely performed when the occurrence of cerebral vasospasm can be anticipated. Furthermore, because the incidence of cerebral vasospasm after ICA closure is far greater in an acute than in an elective treatment situation, we hypothesize that the results of tests to preoperatively evaluate tolerance to elective carotid artery closure are invalid in situations of acute SAH.

If the management of a patient with SAH-induced vasospasms is difficult, the management of a patient with carotid occlusion in the face of acute SAH is doubly so. In addition to standard management for vasospasm and DIND, including control of ICP intravenous administration of nimodipine, aggressive CSF drainage, and aggressive hypertension-hypervolemia-hemodilution (HHH) therapy (systolic blood pressure > 200 mm Hg, central venous pressure > 12 mm Hg, pulmonary capillary wedge pressure > 14 mm Hg), pentothal sedation to burst suppression, and active cooling with muscle relaxation in severe cases, endovascular evaluation and treatment is considered. Furthermore, the cerebral perfusion pressure must be very carefully monitored and kept within quite narrow limits.46

Intraarterial infusion of papaverine has been used in the past, but according to studies by Oskouian et al.35 and Polin et al.,37 no long-term clinical benefit has been demonstrated, although significant improvements in vessel diameter and blood-flow velocity have been shown. Intraarterial infusion of calcium-channel blockers like nimodipine,10,17 verapamil14 or nicardipine5 has also been shown to lead to angiographic improvement in vasospasm, but it is not yet clear whether this leads to clinical improvement, and there is a strong need for a randomized controlled trial.

Transluminal balloon angioplasty, first reported by Zubkov et al.57 in 1983, seems to be more effective than pharmacological treatment,58 but several limiting factors must be considered. Among these factors is timing. Early intervention seems crucial to successful treatment, as demonstrated by Rosenwasser et al.40 These authors showed that results were inferior if treatment commenced more than 2 hours after onset of DIND, findings that prompted an even more aggressive approach of prophylactic TBA in high-risk Fisher Grade 3 patients.30 Nevertheless, in a clinical setting involving sedated, poor-grade SAH patients, detection of vasospasm within 2 hours of onset is very difficult as the patients cannot be tested for DIND, and TCD is not performed continually.
Internal carotid artery trunk aneurysms and SAH

Another limiting factor is vessel size. The smallest TBA balloon diameter available is ~ 2 mm.58 Although this is adequate for many vessels, the main problem site in our patients has been the ACoA or the PCoA. In several of our patients in whom preoperative DS angiography with anatomical evaluation of ACoAs and PCoAs demonstrated favorable cerebral collateral circulation, massive late-onset cerebral infarctions developed when serial TCD measurements demonstrated vasospasm. Thus, the lethal second insult is probably due to the vasospasm strangulating the cerebral collateral blood vessels, such as in the ACoA and ipsilateral PCoA. In addition, MCA vasospasm will further decrease an already lowered regional cerebral blood flow in the MCA perfusion territory. As TBA of the ACoA and PCoA is extremely difficult and risky, it is uncertain whether TBA would be of benefit to this type of patient.

Consequently, we believe our data lend support to the view that if acute ICA sacrifice is performed prior to or within the very first days of the vasospasm phase of a major SAH, it should be accompanied by concomitant bypass surgery. Extracranial–intracranial bypass has been performed successfully in the management of refractory vasospasm after SAH6,26,39 and high-flow EC–IC bypass operations have been performed successfully to prevent stroke in patients with giant aneurysms who cannot tolerate ICA occlusion.38,41,49 Given the large perfusion territory endangered, we believe the bypass ought to be of a high-flow type. Consequently, we hypothesize that high-flow EC–IC bypass, when performed within the time frame prior to the onset of vasospasm, may prevent the vasospasm-induced cerebral infarcts and subsequent deaths associated with acute ICA sacrifice in SAH. If the bypass operation cannot be performed at the hospital where the ICA was sacrificed, the patient could be transferred to an institution in which such surgery is routinely performed.

Conclusions

Internal carotid artery BBAs are rare, small, and difficult to treat endovascularly, with coil placement successfully accomplished in only 2 of 14 patients in this series. These aneurysms rupture easily during surgery (6 of 11 cases). Intraoperative aneurysm rupture invariably led to ICA trap ligation. Sacrifice of the ICA within 48 hours of an SAH led to a very poor outcome, even in patients with adequate collateral capacity on preoperative angiography, probably because of vasospasm-induced compromise of the cerebral collateral vessels. We hypothesize that high-flow EC–IC bypass, when performed within the time frame prior to the onset of vasospasm, may prevent the vasospasm-induced cerebral infarcts and subsequent deaths associated with acute ICA sacrifice in SAH.

Disclaimer

The authors do not have any personal or institutional financial interest in drugs, materials, or devices described in the article.

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Fig. 4. Diagram summarizing the study design and outcomes.
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