The efficacy of endosaccular aneurysm occlusion in alleviating neurological deficits produced by mass effect

VAN V. HALBACH, M.D., RANDALL T. HIGASHIDA, M.D., CHRISTOPHER F. DOWD, M.D., STANLEY L. BARNWELL, M.D., PH.D., KENNETH W. FRASER, M.D., TONY P. SMITH, M.D., GEORGE P. TEITELBAUM, M.D., AND GRANT B. HIESHIMA, M.D.

Departments of Radiology (Neurointerventional Section) and Neurological Surgery, University of California Medical Center, San Francisco, California; and Department of Neurosurgery, Oregon Health Sciences University, Portland, Oregon

Endovascular obliteration of intracranial aneurysms with preservation of the parent artery (endosaccular occlusion) has been advocated for patients who fail or are excluded from surgical clipping and cannot undergo Hunterian ligation therapy. To clarify the effect that endosaccular occlusion has on the presenting neurological signs, 26 patients with aneurysms and symptoms related to mass effect who underwent this therapy were followed for a mean of 60 months. Only patients with objective neurological deficits who had not suffered a hemorrhage were included in this series. Response to therapy was classified into one of three groups: “resolved,” if the patient had complete resolution of presenting signs; “improved,” if significant and sustained improvement was recorded in the neurological examinations, and “unchanged,” if no change was observed.

Thirteen patients (50%) were classified as resolved, 11 (42.3%) as improved, and two (7.7%) as unchanged. A comparison of patients classified as resolved with those who were improved revealed that the former group had less wall calcification (30% vs. 60%) and a shorter duration of symptoms. Patients with neurological sign resolution (62%) were more likely to have totally occluded aneurysms on late follow-up arteriograms than those who had improvement (28%) or were unchanged (0%). This study suggests that endosaccular embolization therapy can improve or alleviate presenting neurological signs unrelated to hemorrhage or distal embolization in the majority of cases.

Key Words • interventional neuroradiology • aneurysm • mass effect • balloon embolization • endosaccular occlusion • endovascular therapy

Endovascular obliteration of an aneurysm with preservation of the parent artery has recently emerged as a therapeutic option for patients who fail or are not considered candidates for surgical clipping procedures or Hunterian ligation.5,12-14,19-24,37 One of the earliest and most persistent concerns regarding this novel therapy revolved around whether an embolic device positioned in an aneurysmal sac might aggravate or fail to alleviate the associated mass effects. Only a few scattered case reports have documented the result of this therapy on mass effect and the reported follow-up period is relatively short.5,23,24,34,37 This study was undertaken to document the effect that endosaccular embolic occlusive devices have on aneurysms that manifest with mass effect alone.

Clinical Material and Methods

Patient Population

The techniques, indications for treatment, and management complications for aneurysms treated at our institution by endovascular procedures have been summarized in prior publications.13,14,17-24 From among the 322 patients harboring intracranial aneurysms who underwent endovascular treatment by our group within a 10-year period, we selected a group in order to evaluate the effects of endosaccular occlusion on the presenting neurological signs. Patients were included in this study if: 1) their presenting symptoms related to mass effect and they had objective neurological signs; 2) radiographic records included plain x-ray films, magnetic resonance (MR) imaging, or computerized tomography (CT), and they had undergone close clinical and radiographic follow-up review; 3) there was preservation of the parent artery with endosaccular occlusion; and 4) the aneurysm had not hemorrhaged. A total of 26 patients fulfilled these criteria and were included in this study. Twenty-three patients were treated by navigating a detachable silicone balloon into the aneurysm, exchanging the contrast material in the balloon for a hardening material, 2-hydroxyethyl methacrylate (HEMA), and detaching the balloon after its contents
were solidified. In the remaining three patients, platinum coils were delivered into the aneurysmal sac (fibered coils in two and electrolytically detachable coils in one). Clinical and radiographic records of these patients were retrospectively reviewed regarding patient age, sex, aneurysm location, duration of signs, dominant neurological signs, and clinical outcome. Radiographic records determined the size of the aneurysm, location, effectiveness of endosaccular occlusion, presence of aneurysm wall calcification, and presence of intraluminal thrombus. Initial attempts to quantify the percentage of intraluminal thrombus present prior to treatment proved unreliable, therefore it was recorded as present or absent.

The percentage of residual aneurysm following treatment was calculated by the following technique. The initial pretherapy and follow-up arteriograms were obtained with 1-cm washers placed on either side of the skull in two orthogonal planes to correct for magnification and permit calculation of the size of the aneurysm lumen. Comparisons between the measured volume of the aneurysmal sac on follow-up arteriograms and the initial pretreatment arteriograms yielded the percentage residual lumen.

Patient age, sex, location of aneurysm, presence of thrombus, aneurysm size, clinical signs and their duration, presence of wall calcification, percent residual filling of aneurysm on long-term follow-up angiography, and follow-up period are summarized for all patients in Table 1. Patients who had complete resolution of presenting clinical signs without developing new signs were classified as “resolved.” Patients who exhibited substantial objective improvement in presenting neurological signs without new signs were classified as “improved.” In two patients there were no changes in objective visual field deficits, and they were classified as “unchanged.”

The mean age at the time of treatment was 49.3 years, with a range from 16 to 73 years. The location of the aneurysm was cavernous in 13 patients, carotid ophthalmic in five, at the internal carotid artery bifurcation in three, and posterior communicating artery in one case each. The size of the aneurysm ranged from 8 to 60 mm, with a mean of 21.6 mm. The duration of clinical signs ranged from 0.5 to 87 months, with a mean of 18 months. The pa-

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), of Aneurysm</th>
<th>Location of Aneurysm</th>
<th>Thrombus</th>
<th>Aneurysm Size (mm)</th>
<th>Duration of Signs (mos)</th>
<th>Presenting Clinical Signs</th>
<th>Wall Calcification</th>
<th>% Residual Filling</th>
<th>Outcome</th>
<th>Follow-Up Period (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>73, F</td>
<td>cavernous</td>
<td>-</td>
<td>20</td>
<td>4</td>
<td>III, IV, V, &amp; VI palsies</td>
<td>-</td>
<td>10</td>
<td>R</td>
<td>106</td>
</tr>
<tr>
<td>2</td>
<td>36, F</td>
<td>cavernous</td>
<td>+</td>
<td>8</td>
<td>24</td>
<td>Horner’s syndrome</td>
<td>-</td>
<td>0</td>
<td>R</td>
<td>98</td>
</tr>
<tr>
<td>3</td>
<td>57, F</td>
<td>CO</td>
<td>-</td>
<td>18</td>
<td>6</td>
<td>decreased visual acuity</td>
<td>-</td>
<td>5</td>
<td>I</td>
<td>77</td>
</tr>
<tr>
<td>4</td>
<td>56, F</td>
<td>cavernous</td>
<td>-</td>
<td>10</td>
<td>1</td>
<td>VI palsy</td>
<td>-</td>
<td>10</td>
<td>R</td>
<td>76</td>
</tr>
<tr>
<td>5</td>
<td>54, F</td>
<td>CO</td>
<td>-</td>
<td>8</td>
<td>1</td>
<td>decreased visual field</td>
<td>?</td>
<td>30</td>
<td>I</td>
<td>75</td>
</tr>
<tr>
<td>6</td>
<td>32, M</td>
<td>1) cavernous</td>
<td>-</td>
<td>12</td>
<td>8</td>
<td>VI palsy</td>
<td>-</td>
<td>0</td>
<td>R</td>
<td>96</td>
</tr>
<tr>
<td>7</td>
<td>61, F</td>
<td>basilar tip</td>
<td>+</td>
<td>30</td>
<td>4</td>
<td>hemiparesis</td>
<td>+</td>
<td>0</td>
<td>R</td>
<td>74</td>
</tr>
<tr>
<td>8</td>
<td>70, F</td>
<td>cavernous</td>
<td>+</td>
<td>30</td>
<td>24</td>
<td>III, V, &amp; VI palsies,</td>
<td>+</td>
<td>5</td>
<td>I</td>
<td>68</td>
</tr>
<tr>
<td>9</td>
<td>18, F</td>
<td>cavernous</td>
<td>+</td>
<td>10</td>
<td>3</td>
<td>V &amp; VI palsies</td>
<td>-</td>
<td>5</td>
<td>R</td>
<td>68</td>
</tr>
<tr>
<td>10</td>
<td>43, F</td>
<td>cavernous</td>
<td>+</td>
<td>30</td>
<td>12</td>
<td>III &amp; VI palsies</td>
<td>+</td>
<td>20</td>
<td>I</td>
<td>68</td>
</tr>
<tr>
<td>11</td>
<td>56, F</td>
<td>ICA bifarc</td>
<td>+</td>
<td>30</td>
<td>87</td>
<td>decreased visual field</td>
<td>+</td>
<td>20</td>
<td>U</td>
<td>60</td>
</tr>
<tr>
<td>12</td>
<td>16, F</td>
<td>ICA bifarc</td>
<td>-</td>
<td>25</td>
<td>3</td>
<td>hemiparesis</td>
<td>-</td>
<td>0</td>
<td>R</td>
<td>60</td>
</tr>
<tr>
<td>13</td>
<td>37, F</td>
<td>cavernous</td>
<td>-</td>
<td>8</td>
<td>0.5</td>
<td>decreased visual acuity</td>
<td>-</td>
<td>0</td>
<td>I</td>
<td>64</td>
</tr>
<tr>
<td>14</td>
<td>27, F</td>
<td>CO</td>
<td>-</td>
<td>9</td>
<td>72</td>
<td>decreased visual acuity</td>
<td>-</td>
<td>0</td>
<td>I</td>
<td>62</td>
</tr>
<tr>
<td>15</td>
<td>42, F</td>
<td>cavernous</td>
<td>-</td>
<td>20</td>
<td>6</td>
<td>V &amp; VI palsies</td>
<td>-</td>
<td>0</td>
<td>R</td>
<td>61</td>
</tr>
<tr>
<td>16</td>
<td>57, M</td>
<td>ACAO</td>
<td>+</td>
<td>50</td>
<td>60</td>
<td>dementia</td>
<td>+</td>
<td>0</td>
<td>I</td>
<td>54</td>
</tr>
<tr>
<td>17</td>
<td>47, F</td>
<td>cavernous</td>
<td>+</td>
<td>30</td>
<td>36</td>
<td>V &amp; VI palsies</td>
<td>+</td>
<td>10</td>
<td>I</td>
<td>52</td>
</tr>
<tr>
<td>18</td>
<td>53, F</td>
<td>CO</td>
<td>+</td>
<td>30</td>
<td>5</td>
<td>decreased visual field</td>
<td>+</td>
<td>5</td>
<td>U</td>
<td>50</td>
</tr>
<tr>
<td>19</td>
<td>49, F</td>
<td>cavernous</td>
<td>+</td>
<td>18</td>
<td>6</td>
<td>III palsy</td>
<td>+</td>
<td>10</td>
<td>R</td>
<td>50</td>
</tr>
<tr>
<td>20</td>
<td>51, F</td>
<td>CO</td>
<td>-</td>
<td>9</td>
<td>4</td>
<td>III palsy</td>
<td>-</td>
<td>0</td>
<td>R</td>
<td>50</td>
</tr>
<tr>
<td>21</td>
<td>45, M</td>
<td>ICA bifarc</td>
<td>+</td>
<td>60</td>
<td>36</td>
<td>hemiparesis</td>
<td>+</td>
<td>90</td>
<td>I</td>
<td>44</td>
</tr>
<tr>
<td>22</td>
<td>50, F</td>
<td>cavernous</td>
<td>-</td>
<td>18</td>
<td>2</td>
<td>VI palsy</td>
<td>-</td>
<td>0</td>
<td>R</td>
<td>42</td>
</tr>
<tr>
<td>23</td>
<td>70, F</td>
<td>PICA</td>
<td>+</td>
<td>30</td>
<td>15</td>
<td>ataxia</td>
<td>+</td>
<td>0</td>
<td>R</td>
<td>36</td>
</tr>
<tr>
<td>24</td>
<td>60, F</td>
<td>PICA</td>
<td>+</td>
<td>12</td>
<td>12</td>
<td>hemiparesis</td>
<td>+</td>
<td>5</td>
<td>I</td>
<td>31</td>
</tr>
<tr>
<td>25</td>
<td>67, M</td>
<td>PCoA</td>
<td>+</td>
<td>30</td>
<td>0.5</td>
<td>III palsy</td>
<td>+</td>
<td>5</td>
<td>R</td>
<td>16</td>
</tr>
<tr>
<td>26</td>
<td>56, F</td>
<td>cavernous</td>
<td>-</td>
<td>15</td>
<td>36</td>
<td>decreased visual field</td>
<td>-</td>
<td>5</td>
<td>I</td>
<td>12</td>
</tr>
</tbody>
</table>

* CO = carotid ophthalmic; ICA bifarc = internal carotid artery bifurcation; ACAO = anterior communicating artery; PICA = posterior inferior cerebellar artery; PCoA = posterior communicating artery; + = present; - = absent; ? = unknown; roman numerals = cranial nerves. Outcome: R = presenting signs resolved; I = presenting signs improved; U = unchanged.
Endosaccular occlusion of intracranial aneurysms

Statistical Analysis

Statistical analysis was performed between the three outcome groups by conducting multiple t-tests using a Bonferroni adjustment. The three groups were analyzed regarding age of patient, aneurysm size, aneurysm wall calcification, duration of clinical signs, percent residual aneurysm filling after therapy, and the presence of intraluminal thrombus.

Results

Twenty-six patients who presented with mass effect and were treated with endosaccular occlusion were followed for a mean duration of 60 months (range 12 to 106 months). Thirteen patients (50%) had complete resolution of presenting neurological signs without the development of new symptoms. Eleven patients (42.3%) were recorded as having significant and sustained improvement in their neurological condition.

Two patients (7.7%) had no objective improvement in their presenting signs of diminished visual field, although one reported subjective improvement. Comparisons between the patients who exhibited complete resolution of signs and those with significant improvement demonstrated several statistically significant differences. The group with resolution of symptoms showed less wall calcification (30% vs. 60%, p < 0.05), a shorter duration of symptoms (6.6 vs. 26.9 months, p < 0.05), and more aneurysms that remained completely thrombosed (62% vs. 28%, p < 0.05). There were no statistically significant differences between the three groups with regard to patient age, aneurysm size, and presence of intraluminal thrombus.

Illustrative Cases

Case 7: Giant Distal Basilar Artery Aneurysm

This 61-year-old woman with a 7-year history of intermittent but progressive dizziness presented with progressive right-sided weakness of 4 months' duration. Physical examination revealed weakness in the right arm and leg, slow fine finger movements in the right hand, and ataxia. A CT scan revealed a 3-cm partially thrombosed aneurysm arising near the basilar bifurcation and low density in the posterior limb of the internal capsule. An MR image disclosed a partially thrombosed giant (30-mm) basilar tip aneurysm with increased signal in the internal capsule (Fig. 1a and b). Subsequent arteriography confirmed a partially thrombosed aneurysm incorporating the left P1 segment of the posterior cerebral artery into the aneurysm's wide neck (Fig. 1c). Two silicone balloons were navigated into the aneurysm, solidified with HEMA, and detached.

The postembolization arteriogram demonstrated complete aneurysm occlusion (Fig. 1d). Three hours after the procedure, the patient noted improved arm and leg strength. Over the next 3 weeks she regained complete strength in the right arm and right leg and resolution of ataxia. Follow-up CT revealed the balloons to be in an excellent position and the lateral, initially thrombosed, segment of the aneurysm reduced in size (Fig. 1e and f). The low density noted on the initial CT scan in the internal capsule was completely resolved. A follow-up arteriogram at 1 year (Fig. 1g) revealed complete aneurysm occlusion. All follow-up examinations have documented complete resolution of ataxia and normal motor strength. At 74 months following balloon embolization, the patient remained neurologically intact.

Case 12: Giant Supraclinoid Carotid Aneurysm

This 16-year-old girl had an 8-year history of episodes of intermittent right hand and arm numbness, which were increasing in frequency at the time of therapy. Over the 3 months prior to admission she had developed weakness and clumsiness in her right hand and increasing headaches. An MR image disclosed a giant partially thrombosed aneurysm, and arteriography (Fig. 2a) revealed a partially thrombosed giant (25-mm) aneurysm arising from the internal carotid artery bifurcation. The middle cerebral artery was occluded and its territory filled via leptomeningeal collateral vessels from the anterior and posterior cerebral arteries. Four silicone balloons were navigated into the aneurysm and detached.

Over the next 2 days there was improvement in the right hand weakness and clumsiness, which had completely resolved by 2 months after the procedure. A 6-month follow-up arteriogram revealed small residual filling of the aneurysm, which was treated with an additional balloon, resulting in complete aneurysm obliteration. A follow-up angiogram 7 months later (Fig. 2b) confirmed complete aneurysm obliteration. At 60 months postembolization, the patient remained neurologically intact without headaches.

Case 16: Giant Anterior Communicating Artery Aneurysm

This 57-year-old man presented with a 5-year history of progressive memory loss and confusion, a 1-year history of increasing seizures, and episodes of loss of consciousness. His progressive cognitive decline had forced him to cease working as a schoolteacher several years prior to treatment. A mental status examination demonstrated slow mentation, with an inability to remember three objects over 5 minutes, reverse three numbers accurately, or count in increments of 7. A CT scan revealed a heavily calcified aneurysm (Fig. 3a) with extensive surrounding edema; MR imaging confirmed that much of the aneurysm contained well-organized lamellated thrombus and there was extensive edema in the surrounding frontal lobe (Fig. 3b). An arteriogram confirmed that the neck of the aneurysm arose from the anterior communicating artery (Fig. 4 left); introduction of a single balloon resulted in complete occlusion (Fig. 4 right). Subsequent MR imaging at 3 months, 1 year (Fig. 3c), and 2 years revealed complete resolution of surrounding edema. The patient has had dramatic improvement in his mental status since treatment and experienced only two seizures in the past 4 years.

J. Neurosurg. / Volume 80 / April, 1994
FIG. 1. Case 7. a: Proton-density spin-echo magnetic resonance (MR) image (TR 2000 msec, TE 35 msec), axial view, adjacent to the basilar tip demonstrating a signal-void area extending into the upper midbrain with adjacent intermediate signal consistent with chronic thrombus. b: T2-weighted MR image (TR 2000 msec, TE 70 msec), axial view, demonstrating increased signal within the posterior limb of the internal capsule. c: Right vertebral arteriogram, Towne's projection, demonstrating a bilobed aneurysm arising from the basilar tip and incorporating the left posterior cerebral artery. d: Angiogram, same injection and projection status as c, obtained following embolization with two detachable silicone balloons demonstrating complete occlusion of the aneurysm. e: Contrast-enhanced computerized tomography (CT) scan obtained 2 months after balloon embolization demonstrating two high-density balloons in unchanged position within the neck of the aneurysm. A low-density thrombosed portion is noted extending laterally to the left. f: Contrast-enhanced CT scan at the same level as e obtained 6 months following balloon embolization demonstrating markedly diminished size within the thrombosed portion of the aneurysm. Sections through the internal capsule demonstrated complete resolution of the low-density process. g: Left vertebral arteriogram obtained at 1-year follow-up review demonstrating persistent occlusion of the basilar tip aneurysm.

Discussion

The primary purpose of this study was to determine the effectiveness of intra-aneurysmal occlusive devices on the presenting symptoms of mass effect produced by the aneurysm. To our knowledge, no prior publication has dealt exclusively with this topic. Comparisons to surgically treated series are difficult; the majority of surgical series that report aneurysms with mass effect deal with giant aneurysms (≥ 25 mm), which comprised 42% of cases in our series. Although there are a few scattered reports of aneurysms that spontaneously decrease in size or occlude, the majority of aneurysms will increase in size. Giant aneurysms comprise 5% to 7% of published series and 13% of unruptured symptomatic aneurysms. The natural history of giant untreated aneurysms is poor; they are associated with up to 80% mortality within several years. Considering only giant aneurysms that were unruptured at presentation, Peerless, et al., reported the 2-year mortality rate to be 62%. Hashimoto and Handa, recording the outcome of untreated symptomatic unruptured aneurysms, concluded that in all cases neurological symptoms continued or slowly progressed over a long course, often resulting in subarachnoid hemorrhage. Untreated patients with giant aneurysms in a series reported by Miyagi, et al., also had no improvement. The currently accepted therapy for symptomatic unruptured aneurysms can be subclassified into two separate categories, deconstructive (Hunterian or parent artery occlusion)
Endosaccular occlusion of intracranial aneurysms

FIG. 2. Case 12. Left internal carotid angiograms, lateral projection. a: Pre-embolization angiogram revealing a giant aneurysm arising from the internal carotid artery bifurcation. There is occlusion of the middle cerebral artery, the territory of which fills via leptomeningeal collateral vessels from the anterior and posterior cerebral arteries. b: Follow-up angiogram obtained 13 months after initial balloon embolization demonstrating complete obliteration of the aneurysm.

and reconstructive (where the parent artery is preserved). Both of these procedures can be accomplished by surgical and endovascular techniques.

Deconstructive procedures, accomplished by surgical ligation of the parent artery, trapping, or ligation of the parent vessel in association with a bypass, have been shown to be effective in alleviating mass effect.8,11,29,30,33,35,39 Endovascular techniques have emerged as a suitable alternative to trapping and proximal ligation procedures.2,5,10,13,14,17,19,21,24,29,30,33,35,39 Strother, et al.,34 recorded the MR imaging changes in nine patients with giant aneurysms treated with balloon occlusion. Following occlusion, seven of these patients had symptoms related to mass and had a decrease in aneurysm size on serial scans with clinical improvement.

Surgical procedures that obliterate flow into the aneurysmal sac with preservation of the parent artery can also improve mass effect symptoms and signs, especially if the aneurysmal sac is removed or decompressed.8,29 Hosobuchi25 described 40 patients with giant aneurysms, the majority presenting with mass effect, who were treated with a variety of surgical procedures including intra-aneurysmal electrothrombosis with copper wire. He reported complete or marked improvement in signs and symptoms in 80% of cases. Interestingly, three patients treated with intraluminal electrothrombosis exhibited subtotal occlusion on follow-up angiography but remain free of symptoms and maintained their immediate postoperative improvement.

A relative paucity of data has been published regarding the efficacy of endosaccular occlusion on symptoms and signs relating to mass effect. Taki, et al.,37 recently reported 13 aneurysms with mass effect treated by endovascular techniques; however, the effect of treatment on neurological signs was recorded in only one patient who had improved visual acuity following therapy. Higashida, et al.,24 reported improved visual acuity in a patient with a giant carotid ophthalmic aneurysm treated with detachable balloons. Dowd, et al.,7 reported improvement in a patient with a giant vertebral artery aneurysm that continued to exhibit symptoms years after surgical wrapping but improved following fibered coil embolization.

In the present series, complete resolution of presenting neurological signs occurred in 13 (50%) of 26 patients. Significant improvement in neurological examination without the development of new signs or symptoms was observed in 11 patients (42%). Only two patients (8%) had symptoms that were unchanged, and no patient was worse in long-term follow-up review.

Our earliest experience with deconstructive procedures (parent artery occlusion) using detachable balloons taught us that aggravation of presenting signs and symptoms was quite common in the immediate post-treatment period and may last from several weeks to months. Our experience also taught us that the severity of aggravation of symptoms and the degree of headache were often proportional to the size of residual lumen of the aneurysm rather than the overall size of the aneurysm. Since the occlusive detachable balloons are often positioned just proximal to the aneurysmal sac, we assume that aggravation of symptoms following parent artery occlusion is produced by the inflammatory effects of thrombus created within the sac. This also seems to be supported by observations of untreated aneurysms by us and others. Heros and Kolluri16 reported massive cerebral edema following thrombosis of two intracranial aneurysms; Whittle, et al.,40 have also observed adjacent cerebral edema surrounding aneurysms that spontaneously thrombosed. We have followed a large number of patients with cavernous aneurysms who were not symptomatic enough or not candidates for endovascular or surgical therapy. These patients were initially evaluated with CT, MR imaging, and angiography, and often remained symptom-free for long durations of time. The abrupt onset of severe clinical signs and symptoms (most often the presence of new cranial neuropathies and unremitting headaches) would prompt our re-evaluation with CT, MR imaging, and angiography. Comparison with the initial studies often revealed the overall aneurysm size to be unchanged; however, new intra-aneurysmal thrombus, often proven recent in nature by CT or MR imaging, was present and adjacent brain edema could sometimes be demonstrated. Some of these symptoms responded dramatically to high-dose glucocorticoids. If the aneurysm were located entirely within the cavernous sinuses and the symptoms were severe, antiplatelet or anticoagulation therapy was often helpful in alleviating acute symptoms.

Our early experience with the placement of detachable balloons within an aneurysmal sac paralleled our experience with parent artery occlusion. Aggravation of symptoms was not uncommon, especially in those patients who presented with symptoms related to mass effect. Strother, et al.,34 who serially followed giant aneurysms treated with parent artery occlusion, often noted a slight increase in mass effect and the development of adjacent brain edema along with aggravation in headaches that was observed beginning 48 hours following treatment. Therefore, it has been our policy...
to utilize short-term (1 to 4 weeks) high-dose glucocorticoids in most patients with giant aneurysms, aneurysms with significant mass effect on CT and MR imaging, or aneurysms exhibiting neurological signs of mass effect in the immediate pre- and posttreatment periods. Patients in this series, therefore, often received high-dose glucocorticoids 1 to 2 days prior to and up to several weeks following treatment. No patient, however, received steroids for more than 1 month posttreatment, and follow-up neurological examinations were obtained for at least 6 to 12 months at regular intervals. The follow-up period, which ranged from 12 to 106 months with a mean of 60 months in this study, should ensure that any changes are secondary to the endovascular treatment rather than the short-term adjunctive use of glucocorticoids. The use of glucocorticoids, however, makes evaluation of the treatment response in the perioperative period difficult.

Glucocorticoids were not used in Cases 7 and 12; both of these patients had dramatic rapid improvement in neurological signs and clinical symptoms. While aneurysm shrinkage was documented on serial CT scans in Case 7, the immediate improvement posttreatment may have been related to diminished transmitted arterial pulsations.

Marked aneurysm shrinkage was observed in several cases in this series (see Case 7). This occurrence seems a plausible mechanism in alleviating mass-effect symptoms but does not occur in every case, especially heavily calcified aneurysms (as in Case 16). Even aneurysms without intraluminal thrombus can undergo subsequent shrinkage following endovascular occlusion. Rarely does the detachable balloon exactly match the configuration of the aneurysmal sac, therefore thrombosis around the balloon must occur subsequent to treatment. Clot retraction and thrombus resorption can then permit subsequent aneurysm shrinkage. Even embolic devices that conform to the shape of the aneurysm, such as the soft electrolytically detachable coils, occupy only up to 30% of the aneurysm volume (Guglielmi, personal communication, 1992).

One initial intent of this study was to measure serial changes in aneurysm size following treatment; however, differences in slice thicknesses, positioning, and quality of follow-up examinations precluded accurate serial measurement. Clearly, some aneurysms (as in Case 7, Fig. 1) were reduced dramatically in size while others (as in Case 16, Fig. 3) were unchanged; however, both patients exhibited improvement in symptoms. Factors other than aneurysm size reduction must play a role in the improvement of neurological signs observed in this series. Edema surrounding a giant aneurysm is not uncommon and resolution of edema following treatment was frequently observed in our series (Figs. 1 and 3). Intraluminal thrombus is commonly observed within these aneurysms (56% of our cases), often with surrounding edema. The thrombus is frequently multilamellated and of various ages. Prior studies have suggested that platelet interaction and deposition occur in some giant aneurysms. It
Endosaccular occlusion of intracranial aneurysms

seems plausible that thrombus formation and resolution could regularly occur in many giant thrombosed aneurysms. The inflammatory response of intraneurysmal thrombus could therefore produce surrounding brain edema, a condition commonly observed following the Hunterian treatment of giant aneurysms. Theoretically, endovascular treatment of an aneurysm with preservation of the parent artery could reduce or eliminate the site for continued thrombus formation. Another obvious cause of symptoms is episodic aneurysm growth. If growth is arrested, the surrounding brain parenchyma and cranial nerves may well accommodate to the mass effect, allowing resolution of the neurological deficit.

Comparing the differences between the large subgroups in our series revealed that a residual neck remnant was present in only 38% of patients who had complete resolution of symptoms compared to 72% of those who had improvement but no resolution of their symptoms. It should be noted that other differences included less wall calcification (30% vs. 60%) and shorter duration of neurological signs (6.6 vs. 26.9 months) in the group with complete resolution of symptoms compared to the group with significant improvement. Despite the fact that in 60% of cases there was an average residual aneurysm filling of 15%, no patient was worse than before treatment. This suggests that even subtotal endovascular obliteration of an aneurysm presenting with mass effect may alter the natural history. However, it is well established that subtotal occlusion of an aneurysm will not eliminate the future risk of bleeding, so every attempt should be made to completely exclude the aneurysmal sac from circulation. Difficult aneurysm anatomy as well as limitations of embolic device shapes (primarily balloons in this series) precluded complete aneurysm occlusion in these patients.

The majority of aneurysms in this series were treated with detachable silicone balloons; only two patients were managed with fibered platinum coils and one with electrolytically detachable coils. Although we have treated 79 aneurysms with electrolytically detachable coils, the follow-up period in the majority was too short to include them in this study. The electrolytically detachable coil has currently supplanted detachable balloons for the treatment of surgically difficult aneurysms. Additional studies are needed to confirm whether similar improvement resulted from use of endovascularly placed electrothrombotic devices.

Conclusions

The results of our series suggest that endosaccular occlusion of an aneurysm presenting with mass effect will improve the neurological signs in the majority of patients. Features which increase the chance that an aneurysm will respond to this therapy include the absence of wall calcification and a shorter duration of neurological signs. Complete endovascular occlusion increases the chances that total resolution of symptoms will occur. The mechanisms for the response to therapy are incompletely understood.

Acknowledgments

We would like to thank John Kucharczyk, M.D., Ph.D., for his statistical analysis and Chellene Wood for word-processing assistance.

References

patients. AJNR 11:633-641, 1990


V. V. Halbach, et al.