Characteristics and surgical treatment of dolichoectatic and fusiform aneurysms

JOHN A. ANSON, M.D., MICHAEL T. LAWTON, M.D., AND ROBERT F. SPETZLER, M.D.

Division of Neurological Surgery, Barrow Neurological Institute, St. Joseph’s Hospital and Medical Center, Phoenix, Arizona; and Division of Neurosurgery, University of New Mexico, Albuquerque, New Mexico

Dolichoectatic and fusiform aneurysms represent a small subset of cerebral aneurysms and are often among the most difficult to treat. A consecutive series of 40 patients with 41 of these two types of aneurysms is presented, including their clinical characteristics and surgical treatments. Common to all aneurysms was the pathological involvement of a length of blood vessel with separate inflow and outflow sites (nonsaccular). However, dolichoectatic aneurysms have markedly different symptoms and surgical treatments depending on their location in either the anterior or posterior circulation. Anterior circulation aneurysms involved the petrous internal carotid artery (ICA) in one, the supraclinoïd ICA in three, the middle cerebral artery in 13, and the anterior cerebral artery in four patients. Posterior circulation aneurysms involved the basilar artery in 13, the vertebral artery in six, and the posterior inferior cerebellar artery in one patient. Various surgical procedures were performed, including direct clipping, trapping with bypass, proximal occlusion, resection with reanastomosis, transposition, aneurysmorrhaphy with thrombectomy, and wrapping. There was no surgical mortality in the patient series, and treatment was effective in many patients. Overall, outcome at late follow up was good (Glasgow Outcome Scale scores 1–2) in 78% of patients. Patients with anterior circulation aneurysms had better outcomes than patients with posterior circulation aneurysms, with good outcomes in 90% and 65% of the cases, respectively. Dolichoectatic and giant serpentine aneurysms may develop from smaller fusiform aneurysms and represent a spectrum of the same pathological entity. Arterial dissection may also play a role in the initial development of these aneurysms.

KEY WORDS • aneurysm • bypass • dissection • dolichoectasia • fusiform • serpentine • subarachnoid hemorrhage

FUSIFORM and dolichoectatic aneurysms are uncommon cerebral aneurysms, which often have clinical presentations and therapeutic considerations that differ from those associated with saccular aneurysms. There appears to be a spectrum of dolichoectatic aneurysms ranging from small fusiform aneurysmal dilations of a single vessel to giant dolichoectatic aneurysms filled largely with thrombus. The latter have also been described as giant serpentine aneurysms. These aneurysms, particularly the large partially thrombosed lesions, often produce clinical symptoms by compression of surrounding neural structures, by distal embolization, or by causing subarachnoid hemorrhage (SAH).

Because these aneurysms lack a definable neck that can be clipped to preserve the parent vessel, they are particularly difficult to treat surgically. Often the vessel of origin is either circumferentially involved by fusiform aneurysmal dilation or completely lost in a large thrombosed mass with distal branches arising from the aneurysm dome. Eliminating the risks of hemorrhage or compression without compromising distal cerebral blood supply in that vascular territory often requires unusual and technically difficult approaches. This report describes the surgical techniques used to treat various fusiform and giant dolichoectatic aneurysms during an 8-year period. Because both the clinical presentations of and the surgical approaches to lesions of the anterior and posterior circulation tended to differ, these two groups are differentiated throughout this report.

Clinical Material and Methods

Patient Population

Between 1986 and 1994, 40 patients (25 males, 15 females) with symptomatic dolichoectatic aneurysms were treated at the Barrow Neurological Institute. The patients ranged in age from 5 to 77 years (mean 45 years). Thirty-eight patients underwent surgery and two were treated medically with antiocoagulation therapy. Pre- and postoperative neurological function was evaluated using the Glasgow Outcome Scale31 (GOS), in which a GOS score of 1 is a good recovery, 2 is moderate disability, 3 is severe disability, 4 is vegetative, and 5 is dead.
Twenty-one aneurysms were located in the anterior circulation (Table 1), and 20 aneurysms were located in the posterior circulation (Table 2); one patient (Case 21) had two symptomatic dolichoectatic aneurysms along the basilar artery and the petrous internal carotid artery (ICA). Among the anterior circulation aneurysms, one involved the petrous ICA, three involved the supraclinoid ICA, 13 involved the middle cerebral artery (MCA), and four involved the anterior cerebral artery (ACA). Among the posterior circulation aneurysms, 13 involved the basilar artery, six involved the vertebral artery, and one involved the posterior inferior cerebellar artery (PICA). Eight patients had a total of 12 asymptomatic saccular aneurysms that were also detected on angiography.

Clinical Presentation

Six patients with anterior circulation aneurysms presented with thromboembolic symptoms (transient ischemic attacks (TIAs) and/or infarction). Three patients presented with subarachnoid hemorrhage (SAH). One patient’s vision diminished progressively due to compression of the optic apparatus by an ACA aneurysm, and two patients had aneurysms detected during evaluation for seizures. Eight patients had asymptomatic lesions identified during evaluation of apparently unrelated complaints. Preoperative neurological deficits were present in eight patients with anterior circulation aneurysms, including three patients with expressive aphasia, four patients with cranial nerve deficits, and six patients with abnormal motor examinations.

Patients with posterior circulation aneurysms most often presented with symptoms of brainstem compression and cerebellar dysfunction (10 patients), including quadriparesis, ataxia, and respiratory distress. There were no incidental, asymptomatic posterior circulation aneurysms. Four patients presented with SAH, and five patients presented with thromboembolic symptoms (including one patient with “locked-in” syndrome secondary to a brainstem infarct). Evaluation for a seizure revealed an aneurysm in one patient. Thirteen patients with posterior circulation aneurysms had neurological deficits, including 10 patients with cranial nerve deficits and eight patients with abnormal motor examinations. Overall, 11 patients had a clear history of systemic hypertension.

Surgical Treatment

Of the anterior circulation lesions, five aneurysms were treated directly with clip reconstruction of the parent ves-

### TABLE 1

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Location</th>
<th>Aneurysm Type</th>
<th>Presentation</th>
<th>Treatment</th>
<th>Complications</th>
<th>GOS Score</th>
<th>Follow Up (yrs)</th>
</tr>
</thead>
</table>
Six aneurysms were resected in conjunction with a revascularization procedure, except for Cases 14 and 19. Three aneurysms (Cases 2, 7, and 11) treated early in this series were wrapped with muslin. The remaining seven patients underwent bypass, with either parent vessel occlusion or aneurysm trapping and are represented by two cases in Figs. 1 and 2.

Of the 20 posterior circulation aneurysms, 18 were treated surgically. Direct clip reconstruction was possible in only two patients with vertebral artery aneurysms. Four giant lower basilar/vertebrobasilar junction aneurysms were treated by varying degrees of thrombectomy, endarterectomy, and aneurysmorhaphy using clips to reconstruct the parent vessel, which is illustrated by Case 22 (Fig. 3). All four aneurysms were exposed via the far-lateral approach and two required hypothermic circulatory arrest. In eight patients, the parent vessel was occluded to alter the blood flow through the aneurysm and promote thrombosis of its lumen. The vessels in five of these eight patients were revascularized. Three cases of vertebral artery dolichoectasia causing lower cranial nerve palsies were treated with mobilization and transposition of the vessels to relieve neural compression. One patient with a small fusiform PICA aneurysm had this lesion excised via primary reanastomosis of the vessel. Two patients with posterior circulation aneurysms (Cases 23 and 29) were treated with anticoagulation therapy.

**TABLE 2**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Location</th>
<th>Aneurysm Type</th>
<th>Presentation</th>
<th>Treatment</th>
<th>Complications</th>
<th>GOS Score</th>
<th>Follow Up (yrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>21</td>
<td>13, F</td>
<td>1. VBJ</td>
<td>giant dolicho</td>
<td>progressive headache, nausea/vomiting, diplopia, ataxia</td>
<td>BA: thrombectomy, aneurysmorhaphy (HCA); ICA: trapping, ICA–MCA bypass</td>
<td>LCND, pneumonia, herpes encephalitis</td>
<td>2</td>
<td>3.0</td>
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<tr>
<td>22</td>
<td>64, M</td>
<td>BA</td>
<td>giant dolicho</td>
<td>progressive hemiparesis, ataxia, diplopia, ventilator dependence; S/P bilateral VA occlusions</td>
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<td>none</td>
<td>5</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>57, M</td>
<td>BA</td>
<td>giant dolicho</td>
<td>progressive hemiparesis, ataxia, diplopia, ventilator dependence; S/P bilateral VA occlusions</td>
<td>anticoagulation</td>
<td>none</td>
<td>3</td>
<td>2.8</td>
<td></td>
</tr>
<tr>
<td>24</td>
<td>70, M</td>
<td>BA</td>
<td>giant dolicho</td>
<td>progressive hemiparesis, ataxia, diplopia, quadruparesis</td>
<td>thrombectomy</td>
<td>none</td>
<td>5</td>
<td>1.3</td>
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<td>25</td>
<td>43, F</td>
<td>BA</td>
<td>giant dolicho</td>
<td>progressive hemiparesis, ataxia, dysarthria</td>
<td>BA constricting snare (HCA); ICH</td>
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<td>45, M</td>
<td>BA</td>
<td>giant serpentine</td>
<td>progressive hemiparesis, ataxia, dysarthria</td>
<td>STA–SCA bypass; proximal balloon occlusion; thrombectomy</td>
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<td>1</td>
<td>1.5</td>
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<td>27</td>
<td>58, M</td>
<td>BA</td>
<td>giant dolicho</td>
<td>progressive hemiparesis, ataxia, diplopia, dysarthria; S/P rt VA occlusion &amp; coils</td>
<td>STA–SCA bypass; distal clip occlusion; thrombectomy</td>
<td>EDH</td>
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<tr>
<td>28</td>
<td>59, M</td>
<td>BA</td>
<td>dolicho</td>
<td>hemianesthesia, dizziness; episodic dizziness, vertigo, syncope</td>
<td>mobilization, transposition</td>
<td>anticoagulation</td>
<td>2</td>
<td>0.8</td>
<td></td>
</tr>
<tr>
<td>29</td>
<td>66, M</td>
<td>BA</td>
<td>dolicho</td>
<td>episodic hemiparesis, hemianesthesia</td>
<td>mobilization, transposition</td>
<td>none</td>
<td>1</td>
<td>1.0</td>
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<td>30</td>
<td>39, M</td>
<td>lt VBJ</td>
<td>dolicho</td>
<td>progressive headache, hemiparesis</td>
<td>BA: thrombectomy, aneurysmorhaphy (HCA), proximal occlusion (distal Lt VA)</td>
<td>LCND, pneumonia</td>
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<td>lt VBJ</td>
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<td>33</td>
<td>63, M</td>
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<td>proximal occlusion (distal Lt VA)</td>
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<td>5, M</td>
<td>lt VA</td>
<td>giant fusiform</td>
<td>SAH</td>
<td>proximal occlusion, PICA–PICA bypass; occlusion at PICA; distal coil occlusion</td>
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<td>1</td>
<td>3.0</td>
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<tr>
<td>35</td>
<td>61, M</td>
<td>lt VA</td>
<td>giant fusiform</td>
<td>episodic paraparesis, facial numbness</td>
<td>mobilization, transposition</td>
<td>none</td>
<td>1</td>
<td>4.8</td>
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<tr>
<td>36</td>
<td>57, M</td>
<td>rt VA</td>
<td>fusiform</td>
<td>seizure</td>
<td>proximal occlusion (rt VA)</td>
<td>none</td>
<td>5</td>
<td>1.5</td>
<td></td>
</tr>
<tr>
<td>37</td>
<td>38, M</td>
<td>rt VA</td>
<td>fusiform</td>
<td>SAH</td>
<td>clip reconstruction, VA</td>
<td>pneumonia</td>
<td>2</td>
<td>4.5</td>
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<tr>
<td>38</td>
<td>38, F</td>
<td>rt VA</td>
<td>giant fusiform</td>
<td>TIA (acute ataxia, crossed hypesthesia)</td>
<td>proximal occlusion (rt VA), OAEAS</td>
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<tr>
<td>39</td>
<td>42, M</td>
<td>rt VA</td>
<td>giant fusiform</td>
<td>SAH</td>
<td>clip reconstruction, VA</td>
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<td>1</td>
<td>4.8</td>
<td></td>
</tr>
<tr>
<td>40</td>
<td>70, M</td>
<td>rt PICA</td>
<td>fusiform</td>
<td>SAH</td>
<td>resection, end-to-side reanastomosis</td>
<td>none</td>
<td>1</td>
<td>3.8</td>
<td></td>
</tr>
</tbody>
</table>

*Abbreviations: BA = basilar artery; dolicho = dolichoectatic; EAS = encephaloarteriosynangiosis; EDH = epidural hematoma; GOS = Glasgow Outcome Scale; HCA = hypothermic circulatory arrest; ICA = internal carotid artery; ICH = intracerebral hematoma; LCND = lower cranial nerve deficit; MCA = middle cerebral artery; OA = occipital artery; PICA = posterior inferior cerebellar artery; SAH = subarachnoid hemorrhage; SCA = superior cerebellar artery; SCV = subclavian vein; STA = superficial temporal artery; S/P = status post; TIA = transient ischemic attack; VA = vertebral artery; VBJ = vertebrobasilar junction.
Surgical Outcome and GOS Scores

There was no surgical mortality in this patient series. Twelve patients (30%) encountered complications. Four patients had postoperative hematomas that required evacuation; two patients had thrombosis of the parent vessel or bypass that required revision; three patients had new lower cranial nerve deficits, and one patient had a cerebrospinal fluid leak requiring a shunt. Other miscellaneous complications included four patients with pneumonia, one with pneumothorax, one with catheter-induced subclavian vein thrombosis, and one with postoperative herpes encephalitis.

For the entire series, the mean follow-up period was 2.8 years. Overall, 23 patients made good recoveries (GOS score 1, 58%), eight had moderate disabilities (GOS score 2, 20%), three had severe disabilities (GOS score 3, 8%), and six died (GOS score 5, 15%). Patients with anterior circulation aneurysms had better outcomes than patients with posterior circulation aneurysms. In the former group, 18 patients (90%) received GOS scores of 1 or 2, and two patients died. The latter group included 13 patients (65%) with scores of 1 or 2, three patients with severe disabilities, and four patients who died.

Three patients were neurologically worse (by one GOS grade) after treatment. At late follow up, one of these patients had returned to his preoperative condition and one patient had improved compared to his preoperative condition. Neurologically, the third patient remained worse with increased lower cranial nerve deficits after aneurysmorrhaphy and died 1.3 years after treatment.

Five other patients in this series died. Two patients (Cases 22 and 27) with giant basilar artery aneurysms, treated previously at other institutions with vertebral artery occlusions, presented to this institution with progressive, severe lower cranial nerve deficits (GOS score 3). They did not improve with further treatment and died of respiratory complications 6 and 9 months later, respectively. Three patients died from an unrelated illness: one patient (Case 7) died from pneumonia 5 years after treatment (GOS score 1); one patient (Case 36) was diagnosed with acute myelogenous leukemia during aneurysm treatment and died from his disease 1.5 years later (GOS score 1); and one patient (Case 2) developed acute renal failure 4 months after treatment and ultimately died from a bleeding gastric ulcer (GOS score 2).

Many patients presented with progressive symptoms and steady neurological deterioration. Importantly, only one patient continued to deteriorate over time. This elderly patient presented with multiple deficits related to brainstem compression by a giant vertebrobasilar aneurysm. His clinical condition was no worse after treatment (occlusion of the distal vertebral artery), but at his 2.5-year follow-up examination he had become more disabled and dependent on others for daily support. In the remaining patients, the neurological course, as reflected in GOS scores, either stabilized (25 patients, 74%) or improved (eight patients, 24%).

Discussion

Incidence and Location

Dolichoectatic and fusiform aneurysms represent a small portion of intracranial aneurysms. Two large autopsy series of aneurysm patients that total more than 16,000 cases revealed only 15 fusiform type aneurysms (< 0.1%).22,28 A more recent large series reviewing 50,000 cerebral angiograms identified only 31 patients with fusiform-type aneurysms (0.06%).63 and another series of 538 vertebrobasilar aneurysms reported by Pia65 identified four fusiform lesions (0.7%).

Classically, these lesions have been thought to affect primarily the vertebrobasilar circulation.10,22,46,57 In our series, however, there was a much larger proportion of anterior circulation lesions, as has been noted previously.28,35,63 In a review by Yu, et al.,63 of 31 patients with ectasia of intracranial arteries, the carotid circulation was involved in 14 patients, the vertebrobasilar system in eight, and both in nine. In Housepian and Poo’s58 large autopsy series, the anterior and posterior circulation were involved equally. A recent review of what are referred to as giant serpentine aneurysms, which may represent an
extreme example of dolichoectatic aneurysms, reported anterior circulation involvement in 26 of 39 patients, including 18 on the MCA. Our series also identified the MCA as the most commonly involved vessel in anterior circulation lesions. The reason for the predominance of MCA lesions is not clear but may be related to the greater length of that vessel.

Carotid ectasia may be more likely than vertebrobasilar lesions to become symptomatic, thereby accounting for their higher representation in clinical series. In contrast, vertebrobasilar lesions often may be asymptomatic and hence not be discovered until autopsy. In a review of autopsies at the Mayo Clinic by Nijensohn, et al., only seven (30%) of 23 patients with vertebrobasilar ectasia were symptomatic.
Clinical Characteristics

Dolichoectatic aneurysms cause a remarkable diversity of clinical symptoms that can be grouped according to three mechanisms: compression, rupture, or ischemia. Compression of surrounding neural structures is the most common cause of symptoms with dolichoectatic aneurysms.14,43,59

One-third of the patients in this series had symptoms referable to compression of either surrounding cerebrum or adjacent cranial nerves. Lower cranial neuropathies secondary to compression are extremely common with vertebrobasilar dolichoectatic aneurysms and were seen in nine of our patients. Trigeminal neuralgia12,16,36,43 and hemifacial spasm16,19,21,32,33,46 have been reported, but facial numbness and weakness can also occur, as seen in this series. Oculomotor paralysis has been described, usually from third nerve compression.3,26,61 Diplopia also reflects involvement of the trochlear66 and abducens nerves.7,35 Visual symptoms may also be caused by compression of the optic nerve or chiasm,2,41 as in Case 17. Other reported compressive cranial nerve syndromes include sensorineural deafness from acoustic nerve compression,7,41 dysarthria and dysphagia,41 multiple lower cranial nerve palsies,18,22,41,43 glossopharyngeal neuralgia,6 supranuclear ophthalmoplegia, and medial longitudinal fasciculus syndromes.6,16,55

Enlarging basilar artery dolichoectatic aneurysms can also produce symptoms by direct compression of the brainstem and cerebellum,4,17,43,47 from direct deformation or from obstruction of the third ventricle or foramen of Monro.1,14,47 Because some patients had no direct outflow obstruction, transmission of arterial pulsations from the ectatic vessel has been proposed to cause a “water-hammer” effect at the foramen of Monro, producing functional obstruction and hydrocephalus.5,17 In these cases the pressure within the dilated lateral ventricles remains low in accordance with Pascal’s law.5,17

Compared to dolichoectatic aneurysms, rupture is more common with giant saccular aneurysms.14,27,58 Subarachnoid hemorrhage occurred in only seven (18%) of the 40 patients in this series, an incidence that is at the low end of the range reported in other patient series (19%–40%).2,22,35,43

Cerebral ischemia, the other major cause of symptoms from dolichoectatic aneurysms,25,35,43,44,51,56,63 occurred in six patients with anterior circulation aneurysms and in four patients with posterior circulation aneurysms. The formation of thrombus within the lumen of these aneurysms causes ischemic symptoms: by directly impairing flow through the vessel lumen;35 by blocking the lumen of perforating vessels originating along the length of the dolichoectasia;25 and by causing embolization to distal vessels.38,56 Brainstem infarction also occurs because of distortion of the paramedian branches as the basilar artery becomes ectatic and elongated.43,51

Surgical Treatment

The surgical approach to dolichoectatic aneurysms often is technically difficult and requires careful planning and execution. Although the specific approach is tailored for each patient based on the aneurysm’s location, size, configuration, and flow characteristics, a few general considerations apply to most patients. Preoperative evaluation must include complete angiographic evaluation of not only the aneurysm but patterns of collateral flow, evidence of intraluminal thrombus, evidence of distal hypoperfusion, and potential vessels for bypass. Magnetic resonance (MR) imaging is particularly helpful for demonstrating the presence of thrombus within larger dolichoectatic aneurysms. Angiography, however, may underestimate the true size of the lesion by showing only the residual filling lumen.

We approached all anterior circulation aneurysms located on the ICA, MCA, or A1 ACA through a standard pterional craniotomy with extensive drilling of the sphenoid wing. Occasionally an orbitozygomatic osteotomy is needed to increase the exposure. Preserving the superficial temporal artery during the skin incision is important in case distal bypass becomes necessary. We approached the three aneurysms on the A1 branch of the ACA via an interhemispheric approach with partial resection of the falx.

We approached the posterior circulation aneurysms through either a large posterior suboccipital craniotomy or, more frequently, through the far-lateral approach (transcondylar),1,2,4,50,55 which includes resection of the posterolateral third of the occipital condyle. This approach provides the best exposure of the intradural vertebral artery and was used for most of the vertebrobasilar lesions. By enlarging the medial extent of the bone opening, the distal PICAs can be exposed for a PICA–PICA bypass, as in Case 34. Occasionally, the transpetrosal approaches may be needed for midbasilar artery aneurysms as was the case in one patient in this series.

All surgeries were performed with electroencephalography and evoked potential monitoring, and barbiturate medications were titrated to the point of electroencephalography burst suppression for cerebral protection.62 In three cases of vertebrobasilar dolichoectatic aneurysms, the extreme size of the aneurysm prevented safe or complete dissection without prolonged interruption of blood flow. These cases were therefore performed under hypothermic circulatory arrest, as reported previously.54 This procedure is particularly useful for giant basilar dolichoectatic lesions because the surgeon can open the aneurysm, perform an internal thrombectomy, and reconstruct the parent vessel during the period of circulatory arrest.

When dolichoectatic aneurysms compress the brainstem, the goal of surgery must be directed toward decompression, either directly or by proximal vessel ligation. Drake15 reported success with proximal occlusion in selected giant posterior circulation aneurysms but has stressed that morbidity and mortality rates may be substantial with this technique. We prefer a direct approach initially and to achieve decompression by thrombectomy of the aneurysm with either clipping or reconstruction of the aneurysm neck.

Our usual technique for aneurysm thrombectomy was...
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to open the aneurysm dome after either local or global circulatory arrest and to remove thrombus with an ultrasonic aspirator when possible. The ultrasonic aspirator is very effective for delicate removal of hardened thrombus, because it does not subject the aneurysm to the traction or manipulation that would be applied by piecemeal removal with cup forceps or pituitary rongeurs. In most cases the softened aneurysm neck could then be clipped or occluded. In Case 22 (Fig. 3), however, an internal lumen was reconstructed using Surgicel and cyanoacrylate adhesive through the opening in the aneurysm dome. The lumen was thereby reconstituted through the space created by thrombus removal, with minimal exposure and manipulation of the basilar dolichoectasia and surrounding structures.

Most fusiform and dolichoectatic aneurysms do not have discrete necks like saccular aneurysms, and direct clipping generally refers to external reconstruction of the parent vessel lumen with clips. Occasionally, clip reconstruction does not obliterate the aneurysm completely and the residual portion can be wrapped with cotton to reinforce the wall.

Trapping and distal bypass were effective, particularly for anterior circulation lesions. Although proximal ICA and MCA aneurysms are typically considered most suitable for this approach using extracranial donor vessels, the use of other intracranial donor vessels permits trapping and bypass of aneurysms in other locations. The distal A1 branch of the ACA can be anastomosed in a side-to-side fashion to provide bilateral distal flow after trapping a more proximal aneurysm. Similarly, the tonsilar loops of the PICa can be anastomosed to provide bilateral flow after occlusion of one proximal vessel. This consideration is important in PICa aneurysms proximal to the choroidal point, in which ligation would interrupt flow to important brainstem and cerebellar branch vessels.

In certain cases fusiform aneurysms can be resected by direct reanastomosis of the proximal and distal ends of the vessel. The length of the involved segment must be limited, with enough redundancy available in the vessel to avoid tension. All four patients in this series treated with this technique had excellent results.

The use of anticoagulation therapy alone also needs to be considered, particularly in patients with symptoms caused by progressive thrombosis or distal embolization. Anticoagulation therapy has been reported to be effective in patients with fusiform aneurysms and ischemic symptoms. In a series of 13 patients with fusiform aneurysms, the seven treated with anticoagulant agents had no recurrent symptoms or hemorrhagic complications during an 18-month period. Similarly, in a study by Yu et al., two of nine patients with ischemic symptoms treated with anticoagulation therapy were alive after 10 years, whereas seven untreated patients died of progressive brainstem ischemia. It has been suggested that all patients with vertebralbasilar fusiform aneurysms be placed on antiplatelet therapy, even those without ischemic symptoms.

Pathophysiological Findings

Dandy probably first stated that cerebral artery dolichoectasia are of arteriosclerotic origin. Later reports also support the theory of arteriosclerotic degeneration of the vascular wall as the initial pathogenic factor in the development of these lesions. Houser and Poole described grossly evident cerebral arteriosclerosis in 10 fusiform aneurysms as part of their 1958 autopsy series of 113 aneurysms. Their findings included intimal thickening and hyalinization, with lipid-laden phagocytes and cholesterol clefts.

Atherosclerosis, however, may not be the only or even most common cause of dolichoectatic aneurysms. These lesions are so rare, despite the prevalence of atherosclerotic vascular disease, that other factors must contribute to their development. Still, atheroma may enhance the effect of a congenital defect of the media.

In the pathological analysis of 34 dolichoectatic ICAs, atherosclerotic disease was not a significant finding. Rather, the media was irregular without atheromatous plaques. Multiple gaps were present, and the width of the internal elastic laminae varied. The thinning of the media was associated with atrophy of the muscle fibers and hyalinization of connective tissue. Although atherosclerosis may cause a vessel to widen by weakening its wall, it does not elongate vessels as observed in the patients in this and other series.

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Atherosclerosis, however, may not be the only or even most common cause of dolichoectatic aneurysms. These lesions are so rare, despite the prevalence of atherosclerotic vascular disease, that other factors must contribute to their development. Still, atheroma may enhance the effect of a congenital defect of the media.

In the pathological analysis of 34 dolichoectatic ICAs, atherosclerotic disease was not a significant finding. Rather, the media was irregular without atheromatous plaques. Multiple gaps were present, and the width of the internal elastic laminae varied. The thinning of the media was associated with atrophy of the muscle fibers and hyalinization of connective tissue. Although atherosclerosis may cause a vessel to widen by weakening its wall, it does not elongate vessels as observed in the patients in this and other series.

The use of anticoagulation therapy alone also needs to be considered, particularly in patients with symptoms caused by progressive thrombosis or distal embolization. Anticoagulation therapy has been reported to be effective in patients with fusiform aneurysms and ischemic symptoms. In a series of 13 patients with fusiform aneurysms, the seven treated with anticoagulant agents had no recurrent symptoms or hemorrhagic complications during an 18-month period. Similarly, in a study by Yu et al., two of nine patients with ischemic symptoms treated with anticoagulation therapy were alive after 10 years, whereas seven untreated patients died of progressive brainstem ischemia. It has been suggested that all patients with vertebralbasilar fusiform aneurysms be placed on antiplatelet therapy, even those without ischemic symptoms.

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and giant serpentine aneurysms may therefore represent a spectrum of nonsaccular aneurysms that share a common gross and microscopic appearance, and these lesions may arise from the same pathophysiological mechanism: intimal disruption from dissection. The uniform presence of intimal defects, a thinned or absent smooth-muscle layer, the frequent absence of atherosclerosis, the occurrence in all age groups, and the gross and MR-imaging appearance of these lesions support this hypothesis.

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Address reprint requests to: Robert F. Spetzler, M.D., Neuroscience Publications, Barrow Neurological Institute, 350 West Thomas Road, Phoenix, Arizona 85013-4496.