Deep central arteriovenous malformations of the brain: the role of endovascular treatment

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Deep, centrally located supratentorial arteriovenous malformations (AVMs) are uncommon lesions associated with considerable difficulty in treatment. The authors report a series of 14 deep central AVMs treated by endovascular methods and examine the present role of endovascular treatment. This treatment used alone resulted in complete obliteration of AVMs in approximately 15% of cases and reduction in 50% to a size permitting treatment by means of radiosurgery. Reversal of previous neurological signs and symptoms occurred in 35.7% of embolized patients. Overall, nearly 80% of patients had either complete obliteration of the lesion, reduction to a size allowing radiosurgical treatment, or reversal of previous neurological deficits. There were treatment complications in 14.3% of the cases. Endovascular treatment methods may make a significant contribution to the therapy of AVMs that have a particularly poor course and are difficult to treat by other means.

KEY WORDS • arteriovenous malformation • thalamus • basal ganglion • embolization • interventional neuroradiology • endovascular therapy

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Cerebral deep central arteriovenous malformations (AVMs) are uncommon lesions associated with considerable difficulty in treatment. The authors report a series of 14 deep central AVMs treated by endovascular methods and examine the present role of endovascular treatment. This treatment used alone resulted in complete obliteration of AVMs in approximately 15% of cases and reduction in 50% to a size permitting treatment by means of radiosurgery. Reversal of previous neurological signs and symptoms occurred in 35.7% of embolized patients. Overall, nearly 80% of patients had either complete obliteration of the lesion, reduction to a size allowing radiosurgical treatment, or reversal of previous neurological deficits. There were treatment complications in 14.3% of the cases. Endovascular treatment methods may make a significant contribution to the therapy of AVMs that have a particularly poor course and are difficult to treat by other means.

Clinical Material and Methods

Selection and History

We reviewed the records of 14 patients with AVMs involving the thalamus, basal ganglia, and/or internal capsule who had been treated by endovascular methods at New York University Medical Center and the Hospital of the University of Pennsylvania during the period from 1988 to 1992. Individuals with aneurysm malformation of the vein of Galen presenting during the neonatal period were excluded because these lesions differ anatomically and clinically. All lesions were symptomatic. Six of the patients had experienced an intracranial hemorrhage. Two had recovered completely from the hemorrhage and were neurologically intact at the time of embolization. The remaining four with a history of hemorrhage manifested static neu-
Embolization of thalamic AVMs

**TABLE 1**

<table>
<thead>
<tr>
<th>Pretreatment Signs &amp; Symptoms*</th>
<th>No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>intracranial hemorrhage†</td>
<td>6</td>
</tr>
<tr>
<td>progressive neurological deficit</td>
<td>8</td>
</tr>
<tr>
<td>hemiparesis</td>
<td>4</td>
</tr>
<tr>
<td>diplopia</td>
<td>1</td>
</tr>
<tr>
<td>memory deficit</td>
<td>1</td>
</tr>
<tr>
<td>developmental delay</td>
<td>1</td>
</tr>
<tr>
<td>sensory deficit/headache</td>
<td>1</td>
</tr>
</tbody>
</table>

* Static neurological deficits unchanged for more than 3 months prior to embolization.
† Of the six patients with intracranial hemorrhage who underwent embolization, four had no residual neurological deficit, and four had static neurological deficits unchanged for more than 3 months.

Neurological signs and symptoms or a history of development of newer embolic agents; therefore patients with developmental delay, each deficit occurring in one patient.

Hemorrhage referable to a deep central AVM (Table 1).

Twelve of the lesions were unilateral with a right-to-left ratio of 1.4:1; two of the lesions were bilateral.

Endovascular treatment methods used after 1988 are significantly improved from those employed prior to that time because of advances in microcatheter technology and development of newer embolic agents; therefore patients treated prior to 1988 were excluded from evaluation. Since 1988 N-butylcyanoacrylate (Avacryl) has been our embolic agent of choice. Prior to 1988 isobutylcyanoacrylate was the acrylic compound used for embolization. Avacryl not only provides permanent vessel occlusion but also confers significant safety advantages over isobutylcyanoacrylate, including more flexibility in injection time.

Surgical Procedure

Percutaneous embolization procedures were performed using variable-stiffness microcatheters which were placed within vessels feeding the AVM nidus. Selective angiographic examination was performed to exclude branches supplying normal brain. If normal vessels were identified, the catheter was repositioned; if it was impossible to eliminate filling of normal branches, the pedicle was not embolized. If no normal vessels were identified on selective angiography, 30 to 40 mg of sodium amytal was injected through the microcatheter, and the patient was examined for the presence of new neurological deficits prior to embolization.

Results

Surgical Treatment

Endovascular embolization alone or in combination with radiosurgery was used to treat 14 deep central AVMs involving the thalamus, basal ganglia, and/or internal capsule. Thirteen of the 14 (92.9%) AVMs were initially too large to undergo radiosurgery. Embolization was performed to reduce the size of the lesion, reduce venous hypertension, and permit radiosurgical treatment whenever possible. Following embolization, complete obliteration of the lesion was achieved in 14.3%. In nearly one-third of patients, regression of neurological deficits occurred within 1 month following partial embolization.

In an additional 50% of the cases, embolization reduced the lesion to a size where radiosurgery was possible and all of these patients have undergone radiosurgery.

Angioarchitecture of the Lesions

The basilar perforating arteries constituted the major supply to these lesions. The thalamoperforating and lenticulostrate arteries were the most common sources of arterial supply and either or both were involved in supplying 13 of the AVMs. Supply from both thalamoperforating and lenticulostrate vessels was present in seven patients. We were able to unequivocally identify the supply from thalamogeniculate branches of the posterior cerebral arteries in only two cases, although overlap of enlarged posterior choroidal vessels in this region may have masked thalamogeniculate supply in other patients.

Three AVMs received perforating artery supply solely from the anterior portion of the circle of Willis via the lenticulostrate arteries. Two AVMs received perforating vessel supply from only the posterior portion of the circle of Willis. The remaining nine lesions received supply from both anterior and posterior portions of the circle of Willis, reflecting the large rostrocaudal extent of most of the lesions (Fig. 1). Some contribution from cortical branches was present in four AVMs (28.6%) but in no case did these vessels constitute the major portion of supply. The deep origin of arterial supply constitutes a major dif-
ficulty in the surgical therapy of these lesions. Arterial supply from both anterior and vertebrobasilar circulations was present in most (64.3%) of the patients. Nearly 15% of the lesions were bilateral.

Angiograms obtained in two patients demonstrated intranidal aneurysms (Fig. 2); however, no feeding artery aneurysms were identified. Late-sequence angiographic films of venous drainage were evaluated in all patients. Deep venous drainage, defined as drainage via either the basal vein of Rosenthal or the internal cerebral vein to the vein of Galen and straight sinus, was present in all patients as expected from the location of the lesion (Fig. 3).

A single embolization procedure was performed in seven patients and the remaining seven were treated in a staged manner with up to four procedures. Individuals under the age of 19 years had embolization performed under general anesthesia, whereas local anesthesia with sedation was used in older patients. Final postembolization films were compared with preembolization films to estimate the percentage of each AVM that had been occluded. It was found that the nidus size of seven lesions (50%) had been reduced to less than 35 mm. All seven patients subsequently underwent radiosurgery (Table 2). The average amount of decrease in nidus size was 67.2% and ranged from 30% to 100%.

One-hundred percent obliteration or complete cure of the lesion was achieved in two (14.3%) additional cases. Complete obliteration was present following a single embolization in one case; however, the patient refused follow-up angiographic evaluation to confirm continued occlusion. In a second case, over 90% occlusion of the nidus was achieved at the time of embolization and follow-up angiograms revealed that the lesion was completely obliterated.

Within 1 month of the first embolization, four patients (28.6%) showed improvement of neurological deficits, including partial resolution of cranial nerve palsy and ophthalmoparesis in one patient and of hemiparesis in three.

**Fig. 1.** Angiograms demonstrating a large arteriovenous malformation (AVM) receiving its arterial supply from both the anterior and posterior portions of the circle of Willis. A and B: Preoperative anteroposterior (A) and lateral (B) left carotid injections. C and D: Anteroposterior (C) and lateral (D) left vertebral injections demonstrating involvement of the right thalamus with extension into the medial parietooccipital region. E and F: Postembolization views of right internal carotid (E) and left vertebral (F) show residual AVM prior to radiosurgery.
At follow-up examination 4 years later, the residual oculomotor disturbance was corrected with strabismus surgery and prisms. Improvement of previous hemiparesis has occurred in both patients who had complete obliteration of their lesions by embolization. The third patient with hemiparesis prior to embolization continues to manifest normal strength and gait with mildly increased deep tendon reflexes 2 years after treatment. A fifth patient experienced resolution of upper-extremity sensory deficits and relief of previously debilitating headaches within 2 weeks after the procedure. Overall, five (35.7%) of the patients manifested improvement in neurological signs or symptoms following embolization, seven (50%) were unchanged neurologically, and two experienced intracranial hemorrhage 3 months and 1 year, respectively, following embolization. In both of these individuals, transient worsening of hemiparesis occurred but subsequent return to neurological baseline was achieved.

Six (42.9%) of the patients had lesions too large for radiosurgical treatment even after embolization; however, following only partial embolization two experienced significant resolution of neurological deficits including hemiparesis and ophthalmoplegia. Their clinical improvement has persisted for 2 and 5 years, respectively, without additional therapy.

New-onset neurological deficits associated with embolization occurred in two patients (14.3%). Worsening of previous left-sided hemiparesis and development of hemisensory deficits and dysesthesia occurred in one individual. A second patient also sustained a new hemisensory deficit with dysesthesia. These findings are compatible with thalamic pain syndrome. In these last two cases, the preceding embolization procedure had involved occlusion of feeding pedicles originating from the proximal posterior cerebral artery on the side contralateral to the deficit. Amytal injection was accomplished in both cases prior to embolization without neurological change. No significant improvement of the hemiparesis has occurred at 5-year follow-up review in the first patient with embolization-related complication; however, considerable resolution of sensory symptoms occurred with only mild dysesthesia present on the left side of the face and body. Sensory symptoms in the second patient have also markedly diminished. No hemorrhages or mortality occurred in association with the embolization procedures.

Discussion

Lesion Size Reduction

Obliteration rates for AVMs in the range of 80% have been obtained with the use of radiosurgery for small
lesions. The recent report of Sisti, et al., highlights the role of microsurgical resection in small AVMs (<3 cm) even in locations previously considered surgically inaccessible. Despite a preponderance of deep AVMs in their series, a 94% rate of complete surgical resection was achieved with a surgical morbidity of 1.5%. The authors note that the size rather than the location of an AVM represents the greater surgical challenge. These findings indicate that the size reduction achieved in 50% of our patients would open the additional possibility of surgical resection following embolization for this group. Considerable controversy exists regarding the respective roles of microsurgery and radiosurgery for small, critically located AVMs. Size reduction using permanent embolization represents a significant advance in therapy for patients with deep-seated AVMs.

All patients had neurological symptoms or signs referable to the lesions. In eight patients (57.1%), the deficits were acute or progressive over the 3 months preceding treatment, and six (42.9%) remained neurologically stable for at least 3 months prior to embolization.

Procedure-related neurological deficits occurred in two (14.3%) of the patients. Both developed a thalamic pain syndrome known to occur from occlusion of vessels supplying the thalamus. The complete syndrome, first described in 1906 by Déjerine and Roussy, involves sensory deficits affecting all primary modalities on one-half of the body. Many patients also experience painful dysesthesia throughout the involved area. The syndrome has been associated with damage of the posterolateral thalamus, a region usually supplied by thalamogeniculoanterior arteries.

Both patients had undergone occlusion of arterial feeding vessels from the proximal posterior cerebral arteries although vessels specifically identifiable as thalamogeniculodorsal could not be found in either case prior to embolization. The absence of response to selective injection of amytal is believed to have resulted from the relatively high flow into the AVM, preventing sufficient amytal perfusion of adjacent vessels. Alternatively, reflex of the embolic agent into adjacent vessels may have occurred following occlusion of the AVM feeding pedicle. In both cases significant resolution of dysesthesia had occurred by follow-up evaluation.

Management difficulties associated with deep central AVMs have resulted in uncertainty regarding the role of embolization in the treatment of patients with these lesions. The present study demonstrates that embolization can significantly contribute to management in nearly 80% of cases. Endovascular therapy may permit radiosurgical or microsurgical therapy of otherwise untreatable lesions, result in remission of neurological deficits, and, in some cases, result in complete obliteration of these AVMs.

Conclusions

Endovascular techniques are a useful addition to the limited therapeutic armamentarium for treating deep central AVMs and should be considered early in the management of these difficult lesions. The application of combined therapy with embolization followed by either microsurgery or radiosurgery represents a significant advance for patients with deep-seated AVMs.

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EmboliZation of thalamic AVMs


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