Embolization and reduction of the "steal" syndrome in cerebral arteriovenous malformations

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The authors report their experience with embolization of unresectable cerebral arteriovenous malformations in 10 patients; seven showed clinical improvement, two no change, and one became worse. Follow-up studies for up to 9 years suggest that partial obliteration of a lesion that shunts blood away from normal brain alleviates the clinical syndrome by enhancing cerebral perfusion. Thus, satisfactory results may be obtained without occlusion of malformations causing symptomatology on the basis of a cerebral steal.

KEY WORDS arteriovenous malformation embolization cerebral ischemia

The treatment of selected cerebral arteriovenous malformations (AVM) by embolization was first described by Luessenhop and Spence. Details of the procedure were reviewed and expanded in 1965. Since that time few reports regarding the subject have appeared. The purpose of this paper is to report and discuss our experience suggesting that AVM's that cause ischemic symptomatology respond best to this mode of therapy.

Materials and Methods

Ten patients, selected from a group of 46 with cerebral AVM, were treated by embolization on the Neurosurgical Service of the University of Washington from June, 1963, through March, 1972. There were six men and four women, and their ages varied from 25 to 48 years at the time of treatment. Four had had previous craniotomies with partial ligation of intracranial feeding vessels, and one a cervical carotid artery ligation. One patient had had repeated episodes of subarachnoid hemorrhage, and two others static deficits and severe headaches prior to their embolization. The rest of the patients showed progressive neurological deterioration that manifested itself in a variety of ways including intractable seizures, progressive focal neurological deficits, and dementia or a combination of these factors. All of these lesions were considered unresectable by conventional techniques because of location or size.

All patients received arteriographic evaluation including bilateral carotid and vertebral artery studies. Angiography was also used to control the embolization procedure. Radiopaque Silastic beads 1 to 5 mm in diameter were used as emboli. The surgical technique described by Luessenhop, et al., was followed except in the most recent case where a retrograde catheterization of the
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TABLE 1
Results of embolization in 10 cases of cerebral AVM

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age, Sex</th>
<th>Mode of Onset</th>
<th>Interval History</th>
<th>Age and Signs at Embolization</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>18 M</td>
<td>focal motor seizures</td>
<td>increasing frequency of seizures with mental changes</td>
<td>32, rt hemi-hypalgesia, hemiparesis, memory deficits, personality change</td>
</tr>
<tr>
<td>2</td>
<td>23 M</td>
<td>generalized seizures</td>
<td>loss of memory, depression</td>
<td>45, lt homonymous hemianopsia, lt hemiparesis &amp; hypalgesia</td>
</tr>
<tr>
<td>3</td>
<td>18 F</td>
<td>personality change</td>
<td>multiple subarachnoid hemorrhage &amp; generalized seizures; craniotomy with ligation of major feeding vessels</td>
<td>28, mental retardation, loss of most cognitive functions</td>
</tr>
<tr>
<td>4</td>
<td>8 F</td>
<td>mental retardation, seizures</td>
<td>developed spastic lt hemiparesis</td>
<td>48, spastic quadriplegia, dementia</td>
</tr>
<tr>
<td>5</td>
<td>16 M</td>
<td>focal motor seizures</td>
<td>generalized seizures, progressive focal seizures of rt face &amp; hand with speech arrest</td>
<td>26, paresis, rt arm, impaired recent memory, slurred speech, rt 7th nerve paresis</td>
</tr>
<tr>
<td>6</td>
<td>18 M</td>
<td>generalized seizures</td>
<td>craniotomy at age 21 with partial excision; postop rt hemiparesis with slow deterioration of mental function</td>
<td>25, flat affect, impairment of intellect, rt hemiparesis, poorly controlled sensory seizures of rt arm</td>
</tr>
<tr>
<td>7</td>
<td>19 M</td>
<td>subarachnoid hemorrhage</td>
<td>multiple subarachnoid hemorrhage, ligation rt internal carotid, progressive mental deterioration, recurrent headaches</td>
<td>33, intellectual impairment, lt hemiparesis, lt homonymous hemianopsia, ataxic gait, dysarthria</td>
</tr>
<tr>
<td>8</td>
<td>30 M</td>
<td>lt hemiparesis</td>
<td>15 yrs after onset sudden increase in lt-sided findings, intracranial ligation of feeding vessels, progressive seizure disorder</td>
<td>45, lt hemiparesis, hypesthesia</td>
</tr>
<tr>
<td>9</td>
<td>32 F</td>
<td>lt hypesthesia</td>
<td>focal lt-sided seizures, progressive paresis of lt arm</td>
<td>43, lt hemiparesis, slurred speech, skull bruit, severe postictal paresis</td>
</tr>
<tr>
<td>10</td>
<td>18 F</td>
<td>focal seizures</td>
<td>intracerebral hemorrhage, lt hemiparesis, headaches</td>
<td>28, lt hemiparesis, intractable headaches</td>
</tr>
</tbody>
</table>

carotid artery was done under local anesthesia.

Results

Clinical histories, results of embolization procedures, and complications for all patients are summarized in Table 1. In seven patients the clinical status was improved, in two unchanged, and in one worse following embolization through the vertebral artery. No operative mortality occurred; complications resulted in lasting neurological deficits only in the last mentioned case. The following more detailed case reports demonstrate some characteristic points to be discussed.
TABLE 1 (continued)

<table>
<thead>
<tr>
<th>Site and Main Blood Supply</th>
<th>Result</th>
<th>Complication</th>
</tr>
</thead>
<tbody>
<tr>
<td>lt frontoparietal, lt pericallosal, lt middle cerebral</td>
<td>decreased seizures, loss of hemiparesis &amp; sensory deficit, improved memory</td>
<td>transient paresis of lt 12th nerve</td>
</tr>
<tr>
<td>rt Sylvian fissure, rt middle cerebral, rt post. cerebral</td>
<td>loss of neurological symptoms &amp; signs, returned to work, 9 yrs postembolization no symptoms but had SAH following rupture of rt internal carotid aneurysm</td>
<td>none</td>
</tr>
<tr>
<td>rt thalamus &amp; periventricular rt vertebral</td>
<td>neurological deterioration, repeated SAH, died</td>
<td>multiple cranial nerve palsies</td>
</tr>
<tr>
<td>lt parietooccipital, lt post. cerebral, lt post. comm.</td>
<td>no change</td>
<td>transient Horner's syndrome</td>
</tr>
<tr>
<td>lt parietal, lt middle cerebral</td>
<td>decrease in focal seizures, increased strength &amp; improved sensation lt arm with further improvement following 2nd procedure, 4 mos post 2nd embolization had SAH from lt internal carotid aneurysm</td>
<td>embolus lodged in rt middle cerebral artery branch, transient dysesthesia lt hand</td>
</tr>
<tr>
<td>lt frontal, both carotids &amp; vertebral via post. comm.</td>
<td>improved mental function, no sensory seizures, reduced weakness rt arm</td>
<td>several emboli to the lungs without sequelae</td>
</tr>
<tr>
<td>rt parietotemporal, lt carotid via ant. comm., vertebral via post. comm.</td>
<td>improved mental function, able to work, loss of ataxia, dysarthria, &amp; field defect, drowned 1 yr postop</td>
<td>none</td>
</tr>
<tr>
<td>rt frontal, rt middle cerebral</td>
<td>AVM not visualized after embolization, transient improvement in function followed by return of symptoms &amp; signs; arteriogram 1 yr postembolization demonstrated filling of AVM; 6 mos later regression of signs with possible spontaneous thrombosis</td>
<td>none</td>
</tr>
<tr>
<td>rt post. frontal, parietal, occipital, rt middle cerebral, rt post. cerebral, lt ant. cerebral</td>
<td>one seizure every 2 mos, return of lt hand function</td>
<td>neck wound infection, increased frequency of seizures early postop</td>
</tr>
<tr>
<td>rt frontal, rt middle cerebral</td>
<td>no change</td>
<td>5 days of retinal ischemia O.D., normal vision now</td>
</tr>
</tbody>
</table>

Characteristic Case Reports

Case 9

This 43-year-old woman noted intermittent numbness of the left hand at the age of 32. Subsequently she experienced focal seizures and progressive weakness involving the left arm. At the time of hospitalization the patient was having one focal seizure a week with severe postictal paresis of the left arm. Examination revealed atrophy of the left arm, left facial palsy, slurred speech and a bruit over the skull. A brain scan showed widespread uptake over the right hemisphere. The major blood supply to the AVM arose from multiple branches of the
right middle cerebral artery (Fig. 1 upper left and right). Additional contributions came from the right anterior and posterior cerebral arteries as well as from the left side via the anterior communicating artery. Six Silastic beads were embolized through the right carotid artery. An intraoperative arteriogram demonstrated that at least two large vessels feeding the malformation had been occluded and normal vessels which had not been evident prior to embolization filled postembolization (Fig. 1 lower left and right); the procedure was stopped when these vessels were seen to be filling. Following surgery the patient had daily seizures, which over a 2-month period decreased to a frequency of one per month. Concomitantly there was improvement in the paresis of the left arm. At the last follow-up 20 months postoperatively, the patient was experiencing a seizure every 2 to 3 months. She had full use of her left hand, speech was improved, and the left facial paresis was not detectable.

Case 2

This 45-year-old man had had generalized seizures since the age of 23. Twelve
Steal syndrome in cerebral AV malformations

years following the onset of symptoms a carotid arteriogram demonstrated an AVM occupying both banks of the right Sylvian fissure. The seizures were well controlled with anticonvulsants; however 2 years prior to admission the patient lost his job because of progressive mental deterioration. He was depressed and had impaired memory for recent events. Incomplete left homonymous hemianopsia and a mild left hemiparesis and hemihypesthesia were noted. Angiographic studies revealed filling of the lesion from multiple small branches of the right middle cerebral artery (Fig. 2 left), the right posterior cerebral artery, and the right anterior cerebral artery via the anterior communicating artery from the left side.

Carotid artery embolization was accomplished by 17 beads ranging from 1 to 3 mm in diameter. The procedure was stopped when it was noted that normal vessels, previously unvisualized, were filling on the intraoperative arteriogram (Fig. 2 right). A postoperative skull film demonstrated emboli scattered throughout the malformation (compare Fig. 3 with Fig. 4 upper right).

Following surgery the hemiparesis improved, the visual deficit regressed, and there was a reversal of memory and personality deficits so that the patient could return to work. For 9 years he remained asymptomatic and then sustained a subarachnoid hemorrhage. An aneurysm of the right internal carotid artery, which had not been seen previously, was demonstrated by angiography. There was no change in the arteriographic configuration of the malformation. The patient has not been operated upon and has made an uneventful recovery.

Fig. 2. Case 2. Left: Preoperative carotid arteriogram demonstrating an AVM within the banks of the right Sylvian fissure and fed by multiple small vessels derived from the right middle cerebral artery. Right: Postembolization carotid arteriogram showing modest reduction in the size of the lesion and increased filling of normal vessels in the surrounding brain (arrows).

Fig. 3. Case 2. Plain skull film showing emboli scattered throughout the AVM. Compare Fig. 4.
Case 5

This 26-year-old man developed focal seizures involving the right hand and face at the age of 16 followed by generalized seizures 6 years later. The major spells were controlled with anticonvulsants, but the focal seizures could not be stopped. At the time of hospitalization he was experiencing five to seven seizures per day; many of these caused speech arrest. Memory of recent events was impaired, and there was right facial weakness, right hemiparesis, impaired sensation in the right arm, and slurred speech.

Angiography demonstrated a left parietal AVM receiving its primary supply from one major branch of the left middle cerebral artery, with minor filling from branches of the left anterior cerebral artery (Fig. 4 upper left). At the first embolization, 12
Steal syndrome in cerebral AV malformations

Silastic beads were embolized through the left carotid artery; 11 of them clustered within the malformation and the other occluded a branch of the right middle cerebral artery (Fig. 4 upper right). The procedure was stopped following the discovery of the aberrant embolus which reached the opposite circulation by retrograde flow down the left carotid and then entered the right common carotid artery because of its anomalous origin from the aorta. The latter variant is fairly common. An electroencephalogram done 3 days following the procedure showed slowing in the right temporal leads compatible with an infarct; 2 weeks later these abnormalities had resolved. The patient complained of dysesthesia in the left hand although no neurological deficits were demonstrable. The frequency and severity of the seizures decreased. Strength improved in the right hand and arm; there were no detectable sensory abnormalities and speech became normal. Postoperatively the patient could still precipitate a seizure by increased physical activity.

It was concluded that another embolization was needed, and this was accomplished using 16 beads. The emboli reached the same region as those in the first procedure, but at least one, and maybe two, moved to another vessel. The patient then complained of numbness in his right hand and the procedure was stopped; the malformation was still clearly visible by arteriography. Figure 4 lower right demonstrates the collection of emboli within the relatively circumscribed area. The abnormal sensation persisted for about 1 week. Three weeks following the second procedure the patient no longer experienced seizures, even with exercise, and retained good function in his right arm. Recently he had a subarachnoid hemorrhage, and an internal carotid artery aneurysm which had not been visualized on previous studies was discovered. He has been treated without surgery and is asymptomatic at this time.

Discussion

Embolization is effective for the treatment of unresectable arteriovenous malformations when the clinical syndrome is due to ischemia of normal brain; in our experience it was not of demonstrable value for patients with static deficits or recurrent subarachnoid hemorrhage. The significant angiographic findings in each of the successfully treated patients were a small decrease in the volume of the AVM and an appreciable increase in the number of demonstrable normal vessels. These findings were associated with objective clinical improvement in motor and sensory deficits, a major decrease in the frequency and severity of seizures, and a reversal of dementia. Our results have been sustained as long as 9 years; this experience is contrary to most data which suggest that only total excision or total embolic obliteration will produce this degree of clinical improvement.

Since our treatment made little change in the mass of the AVM, embolization probably reduces the amount of blood shunted through the AVM by occluding vessels within the lesion and enhancing the perfusion of normal brain sufficiently to ameliorate the clinical findings. Past evidence suggests that AVM's can steal enough blood to cause ischemic damage. The xenon flow technique has recently shown, unequivocally, that blood flow is reduced in areas of brain surrounding an AVM, and following a subtotal embolization blood flow is increased concomitantly with clinical improvement.

The flow dynamics of the abnormal vasculature associated with lesions such as these has not been studied. Drawing from our results we propose that there are pathways of preferential flow within the malformations, and artificial emboli tend to lodge in these vessels. Once these intrinsic vessels, which we postulate have the highest flow rates, are occluded, the shunt away from normal brain is reduced. This cannot be proven, but it is strongly inferred. Embolization of lesions, such as the one seen in Case 5, demonstrate that the emboli have a propensity to terminate in a closely circumscribed area. When an embolus escapes from this field, a normal vessel may be obstructed, as occurred in this case, giving rise to symptoms. There were other instances, exemplified by Cases 2 and 9, in which the emboli scattered throughout the AVM and occluded only a small fraction of
the total vasculature of the lesion, yet produced relief of symptoms. It seems to us that major channels of flow have to be blocked to account for this, particularly since once this is accomplished, ischemic manifestations are abolished for a long period of time.

It is not our intention to suggest that this approach will be satisfactory in all cases. One of the problems that remains, even after successful results with embolization, is the possibility of subarachnoid hemorrhage. In our patients we ascribe this event to rupture from an associated aneurysm, but it is not inconceivable that the AVM bled. Although the coexistence of AVM and aneurysm is well known, what, if anything, the embolization had to do with the appearance of these aneurysms is not clear. The exact role embolization plays in the treatment of AVM is difficult to assess against the wide range of biological variability that these lesions are known to exhibit. We have, for instance, excluded the possibility that thrombosis occurred in Cases 2, 5, and 9, but we suspect that it did occur in at least one patient in this series (Case 8). We must also consider the possibility that the sort of AVM that causes ischemic symptomatology may behave differently from those that bleed. In the Cooperative Study only 18% of the patients with AVM whose presenting symptom was a seizure ever had a subarachnoid hemorrhage. Moreover, five of six patients who benefitted most from this procedure had a seizure as the initial symptom, and none of them ever had a subarachnoid hemorrhage prior to embolization.

References

Steal syndrome in cerebral AV malformations


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