Recanalization of spinal arteriovenous malformations following embolization

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Recently, therapeutic embolization has been advocated as the treatment of choice for arteriovenous malformations (AVM's) of the spine. However, no study has established lasting benefit from this procedure or determined the incidence of recanalization, as occurs with cerebral AVM's. In this study, six patients were followed periodically after complete obliteration of their AVM's by particulate embolization was shown by immediate arteriography. The study group included three men (aged 59 to 72 years) with spinal dural arteriovenous (AV) fistulas and three women (aged 27 to 38 years) with intramedullary glomus-type spinal cord AVM's. The patients were treated by embolization with 100- to 1000-μm diameter polyvinyl alcohol particles. Clinical improvement, most commonly manifesting as increased lower-extremity strength, occurred in all patients after embolization. However, recurrent symptoms, including weakness, numbness, and urinary incontinence, occurred within 2 and 8 months in two of the three patients with dural AV fistulas and within 2 months in two of the three patients with glomus AVM's, prompting radiological reevaluation. Spinal arteriography revealed recanalization of the AV fistulas and spinal AVM's in five patients. Magnetic resonance (MR) imaging demonstrated a signal-void area caused by intramedullary AVM's. This area disappeared after embolic occlusion, but recurred after delayed recanalization, indicating restored flow through the AVM.

Embolization provides only temporary treatment for many spinal AVM's. After embolic occlusion, delayed reassessment with arteriography and/or MR imaging is indicated, particularly if the symptoms persist or recur. Surgical excision of spinal AVM's provides the only therapeutic means to eliminate flow through the AVM permanently in most patients, and should be considered the treatment of choice when feasible.

KEY WORDS • spinal cord • arteriovenous malformation • embolization • polyvinyl alcohol • spinal arteriography

Since the early 1960's, selective spinal arteriography has been used to assess the radiographic anatomy of arteriovenous malformations (AVM's) of the spine. Recent advances in the understanding of these lesions have led to their classification into intradural AVM's (juvenile and glomus AVM's, and direct arteriovenous (AV) fistulas) and dural AV fistulas. Dural AV fistulas display slow flow and produce symptoms by venous congestion of the spinal cord, whereas intradural AVM's are usually high-flow lesions that elicit symptoms by hemorrhage or by ischemia from arterial steal.

Although Elsberg first successfully treated a spinal AVM by surgery in 1914, treatment of spinal AVM's has included embolization since the work of Doppman, et al., Newton and Adams, and more recently by Djindjian, et al., Riché, et al., and Horton, et al. Various biologically inert embolic agents, including dura mater, metallic pellets, muscle fragments, blood clot, Gelfoam, silicone spheres, silicone fluid, isobutyl-2-cyanoacrylate (IBCA), and polyvinyl alcohol (PVA, formerly Ivalon), have been used. Polyvinyl alcohol foam has been employed to embolize lesions of the head, neck, and spine, and is now advocated for treatment of spinal AVM's. However, the long-term efficacy of embolization for spinal AVM's and the incidence of recanalization are unknown. In this study, six patients were evaluated at frequent intervals following embolic occlusion of their spinal AVM's.

Clinical Material and Methods

The clinical records and radiographic studies of patients with spinal AVM's admitted to The Clinical Center, National Institutes of Health (NIH), between 1983 and 1987 were reviewed. This report includes six...
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![Fig. 1. Spinal arteriograms in Case 2. A: Preembolization study showing a dural arteriovenous fistula (arrows) at L-3. B: Study after embolization with 125- to 500-μm polyvinyl alcohol particles showing complete occlusion of the fistula (arrow). C: Study obtained 2 months after embolization showing recanalization of the fistula (arrows).]

patients who received complete obliteration of their AVM by transcatheter embolization. All six patients underwent delayed repeat spinal arteriography. The clinical data were analyzed with respect to epidemiology, clinical presentation, vascular anatomy, size and type of embolization material, adequacy of treatment, therapeutic effect, interval until recurrence of symptoms, and definitive treatment.

The vascular malformations in these patients were of two types. Three patients had dural AV fistulas, characterized by a vascular nidus supplied by dural branches of the intercostal and lumbar arteries and embedded in the proximal nerve root sleeve and the adjacent spinal dura. Three patients with intramedullary AVM's had glomus-type AVM's; that is, a nidus of tightly packed abnormal vessels within the substance of the spinal cord.

Preliminary arteriography was performed in each case to evaluate the feeding and draining vessels. After catheterization of the feeding vessels, contrast material and embolization particles were flow-directed toward the nidus of the lesion. Particles of PVA,* ranging in size from 100 to 1000 μm, were used for embolization in all cases. The particles were impregnated with barium when received from the manufacturer and were suspended in normal saline solution in glass bottles and steam-autoclaved for 30 minutes for sterilization. Standard embolization techniques were utilized in which feeding vessels were subselectively catheterized with a No. 5 or 6 French catheter under fluoroscopic guidance. The procedure was terminated when flow through the nidus of the lesion was eliminated on arteriography. Vital signs and neurological function were monitored during and after the procedure.

After the patient was discharged from the hospital, evaluation in the neurosurgery clinic was scheduled at 3- or 6-month intervals. Radiological evaluation was conducted using spinal arteriography in all patients. Magnetic resonance (MR) imaging of the spinal cord using a 0.26- or 0.5-tesla magnet was performed in five patients.† Two of the three patients with intramedullary spinal AVM's demonstrated by MR imaging underwent delayed repeat MR studies after embolization.

**Results**

Table 1 and Figs. 1 and 2 summarize the clinical and radiographic findings. All three patients with dural AV fistulas were men (aged 59, 61, and 72 years) who had gradually progressive lower-extremity weakness and sensory loss and urinary incontinence (Table 1). In addition, one patient (Case 1) had back pain and another (Case 2) had bowel incontinence. The glomus AVM's were in young women (aged 27, 28, and 38 years), one of whom (Case 4) had subarachnoid hemorrhage, acute paraplegia, and urinary incontinence. Another patient (Case 5) had acute back pain and numb-

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* Polyvinyl alcohol supplied by Unipoint Laboratories, Inc., High Point, North Carolina.

† Magnet manufactured by Picker International, Cleveland, Ohio.
ness and weakness of the legs. The remaining patient with a glomus-type AVM (Case 6) had gradual loss of strength and sensation of the lower extremities, back pain, and bowel and bladder incontinence.

Five of the six patients underwent therapeutic embolization with PVA at the NIH, and the sixth at another institution. Immediate arteriography of all feeding vessels following embolization documented an absence of flow in all AVM's. Before treatment, MR imaging demonstrated a focal intramedullary signal-void area at the site of the AVM in the three patients with intramedullary AVM's. In two of these patients, MR studies performed several hours following embolization demonstrated obliteration of the focal intramedullary signal-void area produced by the AVM (Fig. 2 lower). The nidus was not detected by MR imaging in the patients with dural AV fistulas.

Following treatment, all patients showed clinical improvement (Table 2). However, recurrent symptoms, increasing back pain, increasing lower-extremity weakness and numbness, and increasing urinary incontinence developed within 2 and 8 months in two patients with dural AV fistulas and within 2 months in two patients with glomus-type AVM's of the spinal cord (Table 3). In one patient, neurological improvement stopped 6 months after surgical interruption of the vein (Case 6). In another institution. Immediate arteriography of all feeding vessels following embolization documented an absence of flow in all AVM's. Before treatment, MR imaging demonstrated a focal intramedullary signal-void area at the site of the AVM in the three patients with intramedullary AVM's. In two of these patients, MR studies performed several hours following embolization demonstrated obliteration of the focal intramedullary signal-void area produced by the AVM (Fig. 2 lower). The nidus was not detected by MR imaging in the patients with dural AV fistulas.

Following treatment, all patients showed clinical improvement (Table 2). However, recurrent symptoms, increasing back pain, increasing lower-extremity weakness and numbness, and increasing urinary incontinence developed within 2 and 8 months in two patients with dural AV fistulas and within 2 months in two patients with glomus-type AVM's of the spinal cord (Table 3). In one patient, neurological improvement stopped 6 months after surgical interruption of the vein draining the AV fistula intrathecally. Reestablishment of flow through the dural fistula with medullary venous drainage was documented by arteriography and he underwent embolical interruption of the spinal AV fistula.

Recanalization of the AVM nidus and reestablishment of supply by the previous feeding vessels after embolic occlusion was seen at arteriography in five of the six patients, and by MR imaging in two patients, in whom a reappearance of the signal-void area within the cord on MR imaging confirmed that blood flow was restored through a glomus-type spinal cord AVM. In a third patient (Case 5) with a glomus malformation, recanalization was documented on follow-up MR studies and arteriography, but she did not have clinical progression. This patient had a period of transient improvement after embolization before returning to her baseline neurological state.

One of the patients with a dural AV fistula (Case 3) underwent embolization with PVA and Gelfoam and has had no evidence of residual AVM on repeat arteriography 1 year later. Surgical interruption of the dural AV fistulas, performed in the other two patients, arrested clinical deterioration. The clinical condition of the patients with glomus lesions stabilized without surgical intervention. They are being followed by regular outpatient clinical assessment.

Thus, flow was reestablished through five of six spinal AVM's (two of three dural AV fistulas and three of three glomus-type AVM's) within 1 year of arteriographically documented complete embolic occlusion.

**Discussion**

Due to recent advances in diagnostic imaging such as digital subtraction arteriography and MR imaging, and improved techniques in interventional radiology (such as the development of highly maneuverable catheters permitting embolization via the anterior spinal artery) and embolic materials, embolization of spinal AVM's is used increasingly and is often performed in lieu of surgery. Although each of the new diagnostic techniques has certain advantages and disadvantages compared to conventional spinal arteriography, experience has defined the specific indications for use with each and has demonstrated that selective spinal arteriography with conventional filming is still optimal for treatment planning. Digital subtraction arteriography has the advantage of ease of performance and a reduced dose of contrast material, yet the resolution is inferior compared to conventional filming

Magnetic resonance imaging provides a useful noninvasive technique for detection and localization of intramedullary spinal AVM's, but spinal dural AV fistulas may not be apparent on MR studies and the nidus of the dural AV fistula is seldom visible. Tadavarthy, et al., introduced PVA as an embolic material for the treatment of AVM's in 1975. Since then, PVA has been shown to have several features that result in it being considered an ideal agent for vascular occlusion. It is slowly absorbed and expands to occlude arteries larger than the internal diameter of the delivery catheter.

Animal experiments demonstrate that PVA adheres to the endothelium without intraluminal
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**TABLE 2**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Embolus Material</th>
<th>Particle Size (um)</th>
<th>Vessels Embolized</th>
<th>Therapeutic Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>PVA</td>
<td>250–500</td>
<td>rt T-10</td>
<td>improved leg strength</td>
</tr>
<tr>
<td>2</td>
<td>PVA</td>
<td>125–500</td>
<td>rt L-2, L-3</td>
<td>improved strength, sensation, continence</td>
</tr>
<tr>
<td>3</td>
<td>PVA, Gelfoam</td>
<td>100–500</td>
<td>rt T-1, lt T-4, costocervical trunk</td>
<td>improved strength, continence</td>
</tr>
<tr>
<td>4</td>
<td>PVA</td>
<td>500–1000</td>
<td>rt T-6, T-7, lt T6–9</td>
<td>improved strength, sensation, back pain</td>
</tr>
<tr>
<td>5</td>
<td>PVA</td>
<td>250–500</td>
<td>lt T-10</td>
<td>improved strength, back pain, continence</td>
</tr>
</tbody>
</table>

*AVM = arteriovenous malformation; PVA = polyvinyl alcohol.*

**FIG. 2.** Spinal arteriograms (upper) and magnetic resonance (MR) images (lower) in Case 5.  
Left: Preembolization studies show an intramedullary glomus-type arteriovenous malformation (AVM, arrows) at T-6.  
Center: Complete occlusion of the AVM was obtained by embolization with 250- to 500-μm polyvinyl alcohol particles, with obliteration of the signal-void area on the MR image (arrows).  
Right: Two weeks after embolization there was recanalization of the AVM (black arrows). Recurrence of the signal-void area (white arrow) on the MR image confirmed that blood flow was restored through the AVM.

wedging, suggesting that target vessel occlusion is not flow-rate dependent. It produces an inflammatory reaction, characterized by polymorphonuclear leukocyte infiltration, in the wall of the embolized artery at 2 weeks. These inflammatory changes disappear by 3 months and an adherent, organized, partially calcified thrombus containing PVA is found at 9 months. Several investigators have concluded that PVA serves as a matrix for the ingrowth of fibroblasts, accounting for its relative permanence. This material, initially difficult to handle, is now prepared in several convenient states that can be used for embolization.

Although the results of some animal studies suggest that the effects of PVA embolization may be permanent and that PVA should be effective in permanently occluding most vascular malformations, including spinal AVM's, there has been no long-term assessment of its use in humans with cerebral or spinal AVM's. Of the 13 reports of embolization of spinal AVM's since the initial description, 12 include clinical follow-up data for all patients, but none reported delayed follow-up arteriography in all patients with embolized AVM's or even in half of the treated patients. In the only series which included delayed arteriographic investigation in more than three patients, 10 of 12 patients with "retromedullary" AVM's had recanalization of the AVM nidus. Of the 23 patients reported to have received delayed arteriography following embolic occlusion of a spinal AVM, at least 14 had evidence of recanalization of the AVM. Only one series reported repeating arteriographic investigation at more than 1 year after embolization (Table 4). Although Latchaw and Gold stressed the importance of periodic follow-up arteriography to determine the long-term effects when embolization of lesions of the head, neck, and spine with PVA was used as primary therapy, only three of the eight patients they reported received delayed arteriography. In their report the only patient with a spinal AVM embolized with PVA showed no
recanalization on repeat arteriography at 4 months.\textsuperscript{23} Thérion, \textit{et al.},\textsuperscript{38} obtained follow-up arteriograms in two of five patients after embolic occlusion of intramedullary spinal AVM’s and documented continued occlusion of the nidus of the AVM in these two patients, but the follow-up arteriograms were performed only 1 and 5 months following embolization. The long-term prognosis of embolized spinal AVM’s has yet to be clarified. Our results indicate that the therapeutic effect of embolization is only transient for many spinal dural AV fistulas and AVM’s of the spinal cord.

The nidus of a spinal dural AVM is embedded in the dura (covering the proximal nerve root) and in the adjacent spinal dura. The dural branch of the spinal ramus of the intercostal artery supplies the dural AV fistula.\textsuperscript{27,28} Blood flowing through the dural AV fistula is carried retrograde through the medullary vein to the coronal venous plexus. The absence of valves between the coronal venous plexus and the radial veins of the spinal cord permits transmission of high venous pressure to the cord tissue, which causes myelopathy.\textsuperscript{1,12,20,27,28} The ideal treatment should eliminate venous congestion of the cord by permanent obliteration of the dural AV fistula. In most patients with spinal dural AV fistulas, the vessel supplying the fistula provides no blood supply to the spinal cord.\textsuperscript{25,28,31,36} Since the artery supplying the dural AV fistula usually arises from the aorta separate from the site of origin of vessels which supply the spinal cord, dural AV fistulas can be occluded by transcatheter embolization, and venous congestion of the cord can be eliminated without significant risk of cord injury. This procedure has been performed successfully by Merland, \textit{et al.},\textsuperscript{25} in a large series of patients, although, as cited above, there is insufficient evaluation after treatment to allow conclusions about the permanency of obliteration of the AV fistula. In addition, IBCA, which was used for embolization, is now known to induce malignancies in laboratory animals\textsuperscript{34} and is not available for clinical use in many countries. Of additional concern is that even patients with spinal dural AV fistulas occasionally become paraplegic following embolization, presumably due to retrograde thrombosis of the coronal venous plexus, or to passage of acrylic through the fistula to the coronal venous plexus before polymerization.\textsuperscript{2,25,30} Furthermore, embolization with any material cannot be safely employed when the same segmental artery supplies both the dural AV fistula and the spinal cord, as occurred in four of our 27 patients with spinal dural AV fistulas.\textsuperscript{13,28,33}

Despite early recanalization and clinical recurrence after embolic occlusion with particulate material, as reported here, embolization of spinal AVM’s is usually at least transiently beneficial, as it was in our patients, and should be considered in certain instances. Embolic occlusion during arteriography permits immediate reduction in venous congestion, and thereby permits cord function to recover and, at least temporarily, halts progression of cord damage. In our experience it has been particularly useful in patients with rapidly progressive myelopathy, such as in those previously considered to have the Foix-Alajouanine syndrome.

Because of the temporary efficacy of embolization with particulate material, it is not considered to be the definitive treatment of choice in patients with spinal dural AV fistulas and medullary venous drainage. Surgical excision of the AV fistula, obliteration of the AV fistula combined with interruption of the vein which carries the blood from the dural AV fistula to the coronal venous plexus, or (in patients with common arterial supply to the dural AV fistula and the spinal

![Table 4](image-url)

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Embolization Material</th>
<th>No. Cases Embolized</th>
<th>Complete Occlusion</th>
<th>Delayed Repeat Arteriogram</th>
<th>Interval After Embolization</th>
<th>Cases With Recanalization</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopman, \textit{et al.}, 1968</td>
<td>steel pellets</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Dopman, \textit{et al.}, 1971</td>
<td>pellets, muscle, Gelfoam</td>
<td>7</td>
<td>5</td>
<td>2</td>
<td>2, 4 wks</td>
<td>0</td>
</tr>
<tr>
<td>Djindjian, 1975</td>
<td>NS</td>
<td>34</td>
<td>NS</td>
<td>0</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Djindjian, \textit{et al.}, 1978</td>
<td>Spongol, dura, silicone</td>
<td>12 (dural)</td>
<td>12</td>
<td>≥ 10</td>
<td>2 yrs</td>
<td>10</td>
</tr>
<tr>
<td>Latchaw &amp; Gold, 1979</td>
<td>PVA</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>4 mos</td>
<td>0</td>
</tr>
<tr>
<td>Merland, \textit{et al.}, 1980</td>
<td>microbeads, IBCA</td>
<td>9</td>
<td>9</td>
<td>2</td>
<td>5 days, 2 mos</td>
<td>1</td>
</tr>
<tr>
<td>Riché, \textit{et al.}, 1982</td>
<td>Spongol, dura, IBCA, microbeads, balloon</td>
<td>17</td>
<td>8</td>
<td>1</td>
<td>1 yr</td>
<td>1</td>
</tr>
<tr>
<td>Riché, \textit{et al.}, 1983a</td>
<td>Spongol, dura, IBCA, blood clot, balloon</td>
<td>21</td>
<td>9</td>
<td>0</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Riché, \textit{et al.}, 1983b</td>
<td>balloon</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>5 mos</td>
<td>0</td>
</tr>
<tr>
<td>Berenstein, \textit{et al.}, 1984</td>
<td>IBCA, balloon</td>
<td>12</td>
<td>9</td>
<td>1</td>
<td>6 mos</td>
<td>0</td>
</tr>
<tr>
<td>Scialfa, \textit{et al.}, 1985</td>
<td>dura, IBCA, PVA</td>
<td>12</td>
<td>6</td>
<td>0</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Horton, \textit{et al.}, 1986</td>
<td>PVA</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>1, 3, 4 mos</td>
<td>2</td>
</tr>
<tr>
<td>Thérion, \textit{et al.}, 1986</td>
<td>PVA</td>
<td>5</td>
<td>5</td>
<td>2</td>
<td>1, 5 mos</td>
<td>0</td>
</tr>
<tr>
<td><strong>totals</strong></td>
<td><strong>157</strong></td>
<td><strong>≥ 66</strong></td>
<td><strong>≥ 23</strong></td>
<td><strong>≥ 14</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* AVM = arteriovenous malformation; NS = not stated; PVA = polyvinyl alcohol; IBCA = isobutyl-2-cyanoacrylate.
Recanalization of spinal AVM’s

cord) interruption of the vessel connecting the fistula with the coronal venous plexus is preferred.

The clinical and radiographic findings in patients with intradural spinal AVM’s result from rapid blood flow through the AV nidus.\textsuperscript{28,33} In contrast to dural AV fistulas, in intradural AVM’s the myelopathy results from subarachnoid hemorrhage (the clinical presentation of about 50% of patients with intradural AVM’s is from intradural hemorrhage) and arterial steal.\textsuperscript{28,33} Arterial steal is produced by the combined circumstances of high flow through the AVM and a common origin of the feeding vessels of the AVM with medullary arteries that supply the cord. In this instance sufficient blood flow in the medullary arteries may be diverted away from cord parenchyma into the AV shunt to cause ischemia. The ideal therapy for medullary AVM’s should completely obliterate the AV nidus, via microsurgical excision or embolization, while preserving the blood supply of the cord. Since the major feeding vessels of intradural spinal AVM’s also consistently supply the spinal cord, embolization of these lesions always risks cord injury.

If the juvenile or glomus AVM occupies a considerable portion of the ventral half of the spinal cord, it may be impossible to remove it surgically without incurring unacceptable neurological deficit. Under such circumstances surgical excision should not be attempted. Plans to completely excise or obliterate the malformation may also have to be abandoned intraoperatively if the risk to cord function becomes too great. Reduction of flow in these lesions by embolization may diminish the incidence of subsequent hemorrhage at the nidus of the AVM, despite persistent patency or partial or complete recanalization. One of our patients (Case 4) who had two episodes of intraspinl hemorrhage, each associated with paraplegia and nearly complete sensory loss below T-1, has now been followed for over 2 years after embolization and has had no further hemorrhages, despite recanalization of the AVM and an arteriographic appearance of the AVM similar to that before treatment.

Embollic occlusion may reduce vascular steal and associated ischemia of the cord, and is indicated as primary treatment in certain instances, but delayed reestablishment of flow through the nidus of the AVM usually occurs, and collateral vessels ultimately develop and again supply the AV nidus. Occlusion of intradural spinal AVM’s by selective embolization as therapy preliminary to surgery may also permit safer excision of intradural AVM’s. For intramedullary AVM’s that rechannelize, we repeat embolization only if indicated by recurrent clinical progression.

The prognosis for successful treatment of spinal AVM’s is a function of the preoperative neurological deficit.\textsuperscript{28,33,36} Early recognition and safe treatment, even if it incompletely or only transiently obliterates the AVM, may be beneficial. However, our experience indicates that embolic occlusion of spinal AVM’s is usually not curative, that surgery should be performed in patients in whom it can be carried out safely, and that extended follow-up monitoring will be required to establish the adequacy of embolic treatment of spinal AVM’s.

References


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