Successful excision of a juvenile-type spinal arteriovenous malformation following intraoperative embolization

Case report

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Since the introduction of selective spinal angiography and artificial embolization for the treatment of arteriovenous malformations (AVM's) of the spine, the diagnosis and management of these lesions have rapidly improved. Various biologically inert embolic agents such as dura mater, metallic pellets, muscle fragments, blood clots, Gelfoam, silicone spheres, silicone fluid, isobutyl-2-cyanoacrylate (IBCA), and polyvinyl alcohol (PVA) have been used in the treatment of spinal AVM's by artificial embolization.

In 1989, Hall, et al., reported that in many cases artificial embolization provided only temporary treatment for spinal AVM's and suggested that in most patients surgical excision of these lesions provided the only therapeutic means of permanently eliminating flow through the AVM. Those authors considered surgical excision to be the treatment of choice when feasible. We report here the complete obliteration of a juvenile-type spinal AVM using intraoperative embolization with IBCA combined with surgical resection.

Case Report

This 57-year-old woman was admitted to our institute with a history of progressive paraparesis. In 1975, she had fallen from a height of 1 m and struck her thoracolumbar region. She became paraparetic and developed hypesthesia below the level of the nipples. She had been unable to walk without aid since that time, and the symptoms gradually progressed in a fluctuating manner. In 1988, she became aware of weakness in the right upper extremity. In July, 1989, she was diagnosed as having multiple sclerosis at another hospital and treated with corticosteroids, without improvement. This led to an examination with magnetic resonance (MR) imaging and the diagnosis of a suspected spinal AVM. The patient was admitted to our institute on August 1, 1989.

Examination. No abnormalities were detected on physical examination other than severe neurological deficits. Neurological examination revealed decreased muscle strength in all muscle groups of the right upper extremity (grade 4/5); in both lower extremities, muscle strength was grade 2–3/5 except for the iliopsoas muscle, which was 2/5. The deep-tendon reflexes were exaggerated in all four extremities, with pathological reflexes. Testing for superficial and deep sensation revealed hypesthesia below the level of T-5. There was no remarkable muscular atrophy in the extremities.

Plain thoracic spinal roentgenograms revealed a wid-
Selective spinal angiograms at the right T-7 level demonstrating a large arteriovenous malformation fed by the posterior spinal artery; the draining veins are ascending and descending medullary veins. The left three angiograms are anteroposterior views, and the right two are lateral views.

Preoperative Embolization. On August 7, 1989, artificial embolization with PVA sponges was performed via the femoral route. A catheter was introduced into the right T-7 intercostal artery and, after confirmation of the angiographic appearance of the AVM through the posterior spinal artery, several embolizing PVA sponges (1 x 1 x 10 and 1 x 1 x 40 mm in size) were guided through the catheter. However, all emboli passed through the AVM, so we abandoned this procedure.

Operation. The patient underwent surgery on August 17, 1989. A laminectomy was performed at the C5–T4 levels. All laminae were thin due to the expansive intradural lesions. After removal of the laminae, the dura mater was found to be expanded; the dura mater was opened, and a retromedullary and partially...
Excision of spinal AVM following embolization

Intramedullary AVM was identified. This was fed by the posterior spinal artery and drained into the ascending and descending posterior spinal veins. The extramedullary part of the nidus of the AVM and the ascending posterior spinal vein were noted to be compressing the spinal cord (Fig. 3).

With the aid of an operating microscope, the posterior spinal artery was isolated and cannulated; 1 ml of IBCA was injected through the cannula to embolize the AVM. Immediately after embolization, the wall of the nidus became white, which implied polymerization of the injected IBCA in the nidus. The nidus wall became very hard and blood flow in the draining veins slowed after embolization. Thereafter, the descending draining vein was isolated, coagulated with the bipolar coagulator, and divided. The nidus was isolated in stages from the dorsal surface of the spinal cord, and the extramedullary component of the nidus was completely resected. The residual component of the nidus was thought to be located in the spinal cord and at the surface of the cut nidus. Polymerized IBCA was found intravascularly (Fig. 3). The incision was closed in the usual manner.

Postoperative Course. Following the surgical procedures, the patient initially suffered an increase in weakness in the lower extremities (grade 1–2/5); there-

Fig. 3. Intraoperative photographs showing a large spinal arteriovenous malformation (AVM), of which the nidus (single arrow) is mainly located in the retromedullary portion. Left: Large (double arrow) and small (single arrowhead) draining veins were ascending and descending, respectively. The posterior spinal artery (double arrowhead) feeds the AVM. Center: After resection of the retromedullary portion of the AVM, hardening of the residual intramedullary nidus was confirmed (arrow). Right: Enlarged photograph at the T-1 level demonstrating the occluded intramedullary AVM with intravascular polymerized isobutyl-2-cyanoacrylate (the whitish areas indicated by arrows).

after, signs and symptoms gradually improved and 2 months after the operation she could stand on a tilting table for about 15 minutes. The slowly progressive weakness in the right upper extremity was arrested by the operation. The AVM had disappeared on postoperative spinal angiography 2 months after the operation and there was preservation of the anterior spinal artery originating from the left T-8 intercostal artery (Fig. 4).

This patient does not reside near Osaka and did not want to interrupt her rehabilitation, so we were not able to perform follow-up angiography. Her neurological manifestations gradually improved, however, and 1 year postoperatively she could stand and walk a few meters with the aid of parallel bars.

Discussion

Terminology

In 1977, Kendall and Logue11 first distinguished two types of spinal AVM's angiographically: dural and intradural. Ten years later, Rosenblum, et al.,22 classified spinal AVM's into: dural arteriovenous fistula, glomus-type intradural AVM, juvenile-type intradural AVM, and intradural arteriovenous fistula.

Juvenile-type AVM's of the spinal cord occur most frequently in adolescents and young adults. These AVM's are characterized by their large size, rapid blood flow, and the presence of multiple feeders.43 They are usually located at the cervical level, and surgical removal is quite difficult.
**Embolization Techniques**

The management of juvenile-type AVM's has usually consisted of partial embolization and/or ligation of the feeding arteries.\(^5\)\(^6\)\(^9\) Due to improved techniques in interventional radiology and the use of embolic materials, artificial embolization of spinal AVM's is being used with increased frequency; at present, embolic therapy is thought to be the first choice for treatment of spinal AVM's. Tadavarthy, et al.,\(^24\) reported the clinical value of PVA as an embolic material for the treatment of spinal AVM's in 1975.

In this case, the attempt to embolize the spinal AVM was unsuccessful and the emobolized PVA sponges passed through the AVM. Therefore, we decided to excise the AVM together with intraoperative artificial embolization using IBCA. Alkyl-alpha-cyanoacrylate monomers are low-viscosity liquids. The speed of polymerization is inversely related to the alkyl chain length.\(^15\) Polymerization of IBCA is instantaneous in blood and does not occur in 5% dextrose.\(^22\) Three factors influence the level of obstruction, including polymerization time, injection rate, and blood flow. Slow injection of IBCA at 0.3 ml/sec can obliterate the vessel just distal to the injection site, whereas a 0.3 ml/0.5 sec injection permits the fragmented emboli to enter the small arteries and occasionally a vein.\(^9\)

Riché, et al.,\(^20\) reported the artificial embolization of 21 spinal AVM's and reviewed their clinical experience with IBCA in two patients. Triplegia occurred in one patient with an intramedullary AVM treated with IBCA embolization; 24 hours later, the patient died. However, Merland, et al.,\(^3\) reported the clinical efficacy of IBCA in the treatment of patients with radiculomeningeal AVM (arteriovenous fistula). Scialfa, et al.,\(^23\) also reported four cases of radiculomeningeal AVM (arteriovenous fistula) and one case of extradural AVM successfully treated with this agent. In all cases treated with IBCA, the agent was injected via catheter. In our patient, the feeding arteries included the anterior spinal artery; as the distance between the nidus of the AVM and the tip of the catheter was too widely separated, it was thought to be dangerous to perform embolization with IBCA via catheter.

Generally, management of juvenile-type spinal AVM has consisted of partial embolization and ligation of the feeding vessels. However, Hall, et al.,\(^10\) reported the high incidence (83.8%) of recanalization of spinal AVM's following artificial embolization. Morgan and Marsh\(^17\) also reported that artificial embolization with PVA and/or microfibrillar collagen achieves early clinical improvement with apparent obliteration of the dural AVM, but for the majority of patients this success is not sustained. It should be remembered that recanalization and new collaterals to the AVM develop after proximal embolization (that is, proximal to or at the radicular artery in the treatment of dural or intradural AVM's).

**Conclusions**

In this patient, we planned surgical excision and intraoperative embolization of the nidus itself with IBCA. Embolization permitted us to remove the spinal AVM easily without much bleeding; the residual nidus was considered to have been successfully embolized by the injected IBCA since the AVM was not visible on postoperative selective spinal angiography. It was thought that the embolization of the AVM was successful because the juvenile-type AVM mainly existed dorsal to the spinal cord and the main feeder was the posterior spinal artery.

**References**

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