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SPINAL GIANT INTRADURAL PERIMEDULLARY ARTERIOVENOUS FISTULA: CLINICAL AND NEURORADIOLOGICAL STUDY IN ONE CASE WITH REVIEW OF LITERATURE

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BACKGROUND
Giant intradural perimedullary arteriovenous fistula with massive spinal cord compression is rare. The therapeutic difficulties include whether endovascular embolization or direct surgical excision should be selected. We present a patient with the largest giant spinal intradural perimedullary arteriovenous fistula shown by magnetic resonance imaging so far reported, who was successfully treated by a combination of endovascular embolization and direct surgery.

CASE DESCRIPTION
A 16-year-old girl presented with a giant intradural arteriovenous fistula (perimedullary Type II) at the C4-5 level, manifesting as progressive cervical myeloradiculopathy. The single-hole fistula was supplied by the anterior spinal artery and an ascending artery arising from both the costocervical and highest intercostal arteries with a rapid transit time, and drained superiorly to the foramen magnum, and inferiorly to the thoracic spinal canal, through a huge venous lake at the site of the arteriovenous connection. The patient was treated by transarterial embolization with platinum coils and silk, followed by surgical excision with excellent results at 12 months' follow-up.

CONCLUSIONS
We recommend that such a huge perimedullary arteriovenous fistula with a rapid transit time, and severe cord and root compression, should be treated with embolization followed by surgical excision.

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KEY WORDS
Spinal arteriovenous fistula, intradural perimedullary arteriovenous fistula, spinal arteriovenous malformation, embolization, giant venous lake.

Intradural or perimedullary arteriovenous fistulas, as the name accurately suggests, are abnormal connections between the medullary arteries and veins, without an intervening nidus or glomus [6,8,10,17]. These fistulas have only recently been recognized as distinct from the glomus and juvenile types of intramedullary arteriovenous malformations [4,10,13,14,16,17] and are now considered as a new category [9].

Riche et al [14,16] and Gueguen et al [6] subclassified perimedullary fistulas into three distinct types, based on their angioarchitecture. Type I fistulas are small, with a single feeder, slow transit time, and moderate venous enlargement. Surgical clipping of the fistula site is indicated [6,8,10]. Type II fistulas are single-hole fistulas, supplied by one or two medullary (spinal) arteries, have a more rapid transit time, and often have a venous pocket at the site of the arteriovenous fistula [6,8,10]. Type III fistulas are giant connections supplied by multiple medullary arteries of large caliber that exhibit rapid arteriovenous shunting and drain into greatly dilated and often dysplastic draining veins [6,8,10]. The surgical [4,8,17] and endovascular [5,8,10,13–15] therapy of perimedullary fistula has been described and therapeutic difficulties discussed [7–10,17].

Here we describe the combined endovascular and surgical treatment of a Type II giant intradural

arteriovenous fistula, which achieved considerable clinical improvement. To our knowledge, this is the largest giant intradural arteriovenous fistula shown by magnetic resonance imaging (MRI) so far reported.

CASE REPORT

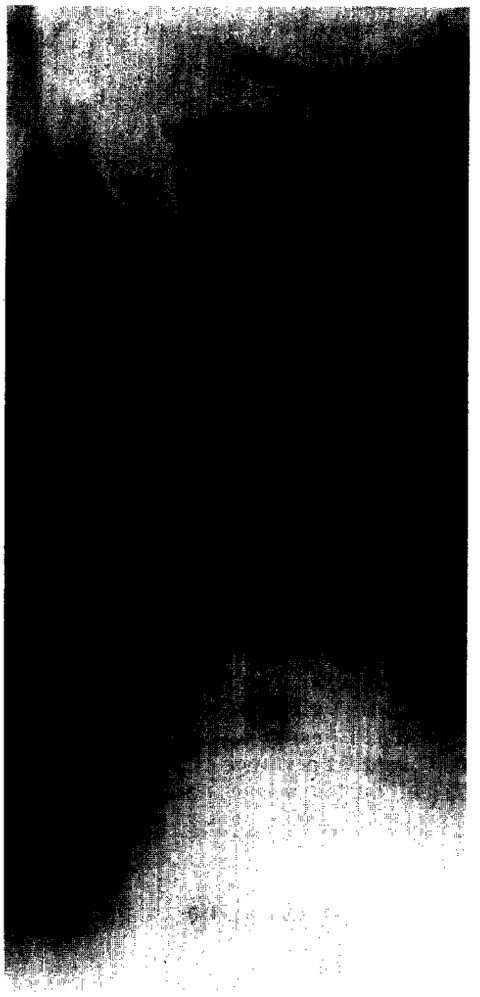
This 16-year-old high-school girl had a history of occasional neck pain aggravated by sneezing or coughing since 13 years of age. She had been doing well at school until 4 months prior to admission when she noticed numbness and weakness of the right hand with occasional pain radiating from the right shoulder to the whole right arm on sneezing or coughing. Three months prior to admission, she noticed slight difficulty in walking. One month before admission, she suddenly developed a clonic convulsive seizure limited to the right leg, lasting for about 1 minute and simulating the "spinal epilepsy" of Penfield [12]. Rapid progression to complete paraplegia occurred over the next 20 days. She was admitted to the Department of Neurosurgery, Saitama Medical School Hospital, after referral from the Department of Neurology, with a tentative diagnosis of spinal arteriovenous malformation in the cervical cord. Her past and family history were noncontributory except for bronchial asthma since she was 10 years old.

EXAMINATION

On admission on May 24, 1994, she had spastic tetraparesis with complete paralysis of the lower extremities. Paralysis of the arms was greatest in the distal muscles. Both hands showed a claw-like appearance with absence of finger extension and restricted finger flexion to about 30% of the normal range of motion. All sensory modalities were lost below the C-5 dermatome. Her neck movement was restricted due to severe pain radiating from the right posterior neck to the right arm, elicited by minimal neck movement, coughing, or sneezing. There was no evidence of Cobb syndrome [3] or Rendu-Osler-Weber syndrome. Auscultation over the head, neck, and shoulder discovered no bruit.

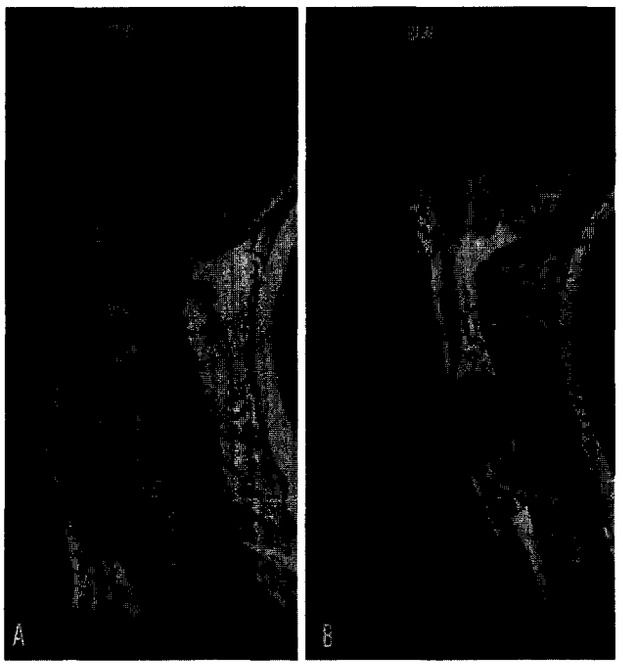
Lateral X-ray films taken with a focus-film distance of 150 cm (Figure 1) showed an enlarged cervical canal with anteroposterior diameters of 20 mm at C-1, 19 mm at C-2, 18 mm at C-3, 21 mm at C-4, 23 mm at C-5, 22 mm at C-6, and 20 mm at C-7. Scalloping was present on the posterior margin of the C-4, C-5, and C-6 vertebral bodies.

MRI revealed a large intradural signal void area, consistent with a huge venous lake with multiple dilated vessels above and below the lake. The cylindrical-shaped venous lake, 40 × 20 × 20 mm, had caused bone erosion of the vertebral bodies and displaced the cord and roots so severely that the cord substance was extremely thin, less than 1-mm thick at C-5 on the midsagittal section (Figure 2). No signal voids within the cord parenchyma cephalad and caudad to C-5 were observed.



1 Plain cervical spine X ray showing the enlarged spinal canal with A-P diameters of 20 mm at C-1, 19 mm at C-2, 18 mm at C-3, 21 mm at C-4, 23 mm at C-5, 22 mm at C-6, and 20 mm at C-7.

Vertebral and selective spinal arteriography revealed a single-hole fistula at C4-5 level, supplied by the anterior spinal artery (Figure 3) and an ascending artery arising from both the costocervical artery and the highest intercostal artery, and draining into a giant venous lake (Figure 4). The arteriovenous shunting was rapid. Dilated tortuous drainers extended caudad to the thoracic spinal canal and cephalad to the foramen magnum (Figure 4). Transarterial endovascular embolization was performed as the first step in the treatment procedures.



2 Preoperative midsagittal T_1-weighted (TR/TE = 500/40 msec) spin echo (**A**), and T_2-weighted (TR/TE = 4500/103 msec) fast spin echo (**B**) MRIs showing signal void areas of a huge cyst compressing the cord and adjacent dilated vessels in the enlarged cervical canal.

ENDOVASCULAR SURGERY (JUNE 15, 1994)

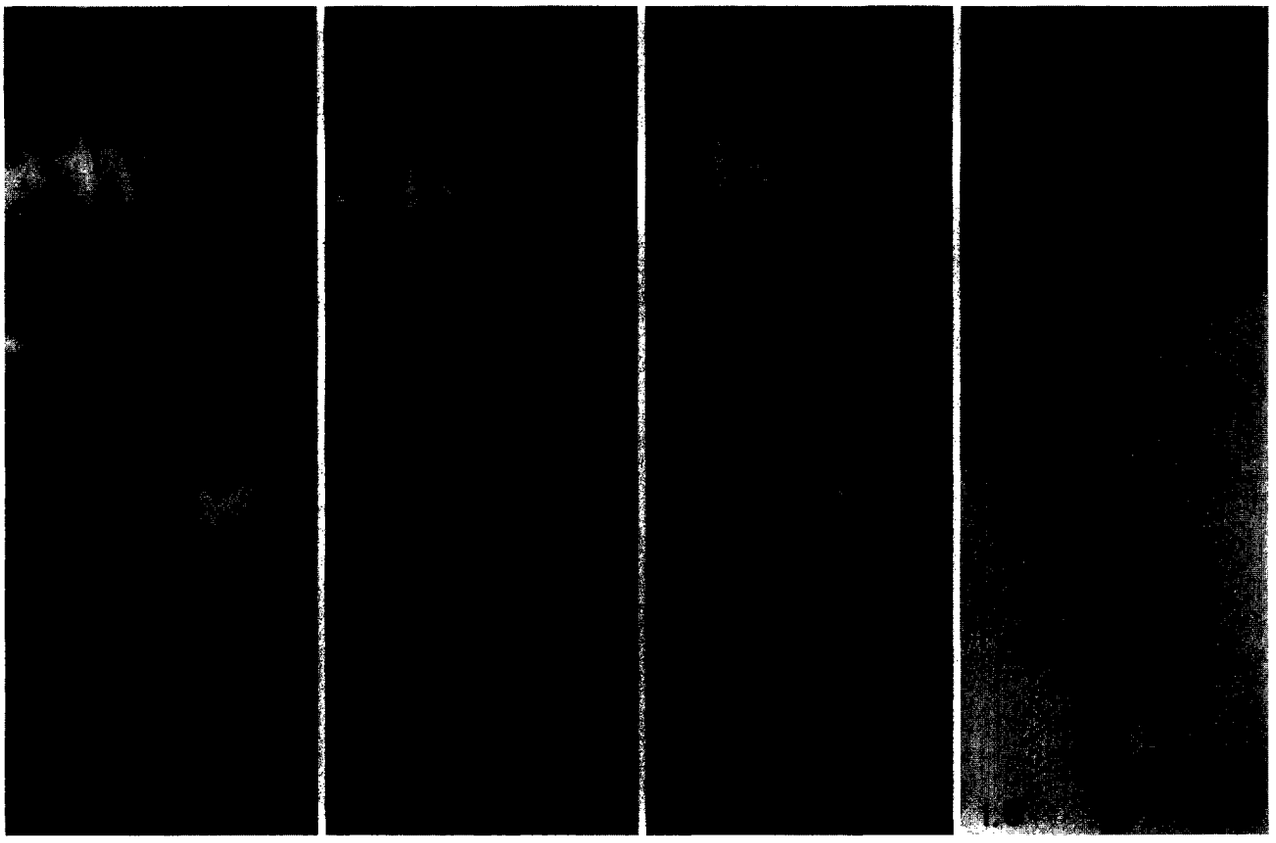
A 5-French catheter was introduced into the highest intercostal artery via the right femoral artery to permit the passage of a Tracker-18 catheter (2.8 French). The microcatheter was navigated into the anterior spinal artery and three platinum coils were detached at the fistula site and in the adjacent proximal and distal feeders. Five silk sutures were injected into the proximal artery with saline to intertwine with the coils to promote thrombus formation. The Tracker-18 catheter was then withdrawn. Selective angiography of the left highest intercostal artery injection through a 5-French catheter opacified neither the fistula nor the venous lake (Figure 4 **E**).

The patient made a dramatic recovery during the following 20 days. The morning after the operation, finger flexion and extension had returned to normal, and her neck-shoulder pain had disappeared. She began to stand and walk on the 5th postoperative day. Selective spinal arteriography on the 15th postoperative day revealed no opacification of the fistula or the venous lake. MRI on the 20th postoperative day (Figure 5) showed disappearance of the signal voids and thrombus formation in the venous lake, but without any reduction in size. At the same time, her symptoms began to return to the preembolization status with an additional symptom not previously described [1,2,8,10,11,14,17]—stiff and painful neck with no tolerance to the jugular compression test due to abrupt increase of neck-shoulder pain extending to the right arm with an unbearable pins-and-needles sensation in the right hand and fingers, associated with leg weakness. This new symptom was probably due to altered hemodynamics with complete loss of arterial inflow into the venous lake but with intact venous drainage. The thrombosed venous lake formed a hardened mass on the compromised cord and may have enhanced reversal to the tetraparesis.

Direct surgery was performed on the 33rd postoperative day.

MICROSURGICAL EXCISION (JULY 18, 1994)

Excision of the fistula, adjacent feeders, and the venous lake was performed through a laminoplastic opening from C-3 to C-7 with the patient placed prone on two blanket rolls. To lower the venous pressure, her head was fixed in a pin headrest while maintaining the anterior chest 10 cm above the operating table, and the head of the table tilted up at 10°. The excised C-4 to C-7 spinal processes were used as the grafts for laminoplasty. Lateral gutters along the laminoarticular line were made on both sides from C-4 to C-7. The laminal arches from C-7 to C-4 were drilled longitudinally, split along the midline, and the split laminae were bent and fixed to the laterally retracted muscles. The exposed dural tube was blue and bulged out. C-3 laminectomy was added. Incision of the arachnoid below the venous lake caused a jet of xanthochromic cerebrospinal fluid 10 cm high to escape. The exposed venous lake was 4.0 × 2.5 × 2.5 cm, shaped like a gourd, blue with whitish band zone, firm, and without pulsatile motion. No blood was aspirated. The venous lake was opened and old blood clots with mural thrombus evacuated, resulting in shrinkage. Underneath the lake, the flattened cord and stretched posterior rootlets were seen through the pia. The anterior spinal artery containing the platinum coils, the ascending artery, and the fistulous vessel connecting with the venous lake were identified. The collapsed fistulous vessel with an external diameter of 2 mm and a length of 1 mm had a T-shaped origin at the junction of the anterior spinal artery and the ascending artery. Bipolar coagulation of the fistulous vessel and the adjacent feeders was followed by excision. The venous lake was then excised except for the ventral wall, which contained numerous, tortuous, small red perimedullary vessels, appar-



3 Left vertebral arteriograms. **A, B, C** are anteroposterior views taken at 0.5, 1.0, and 2.0 seconds after injection. The anterior spinal artery (*large and small arrows*), arising near the termination of the left vertebral artery and proximal to the origin of the posterior inferior cerebellar artery, descends to the venous lake. **D** is a lateral view taken at 1.0 second after injection. *Arrows* indicate the anterior spinal artery, and the *white arrowhead* shows the fistula.

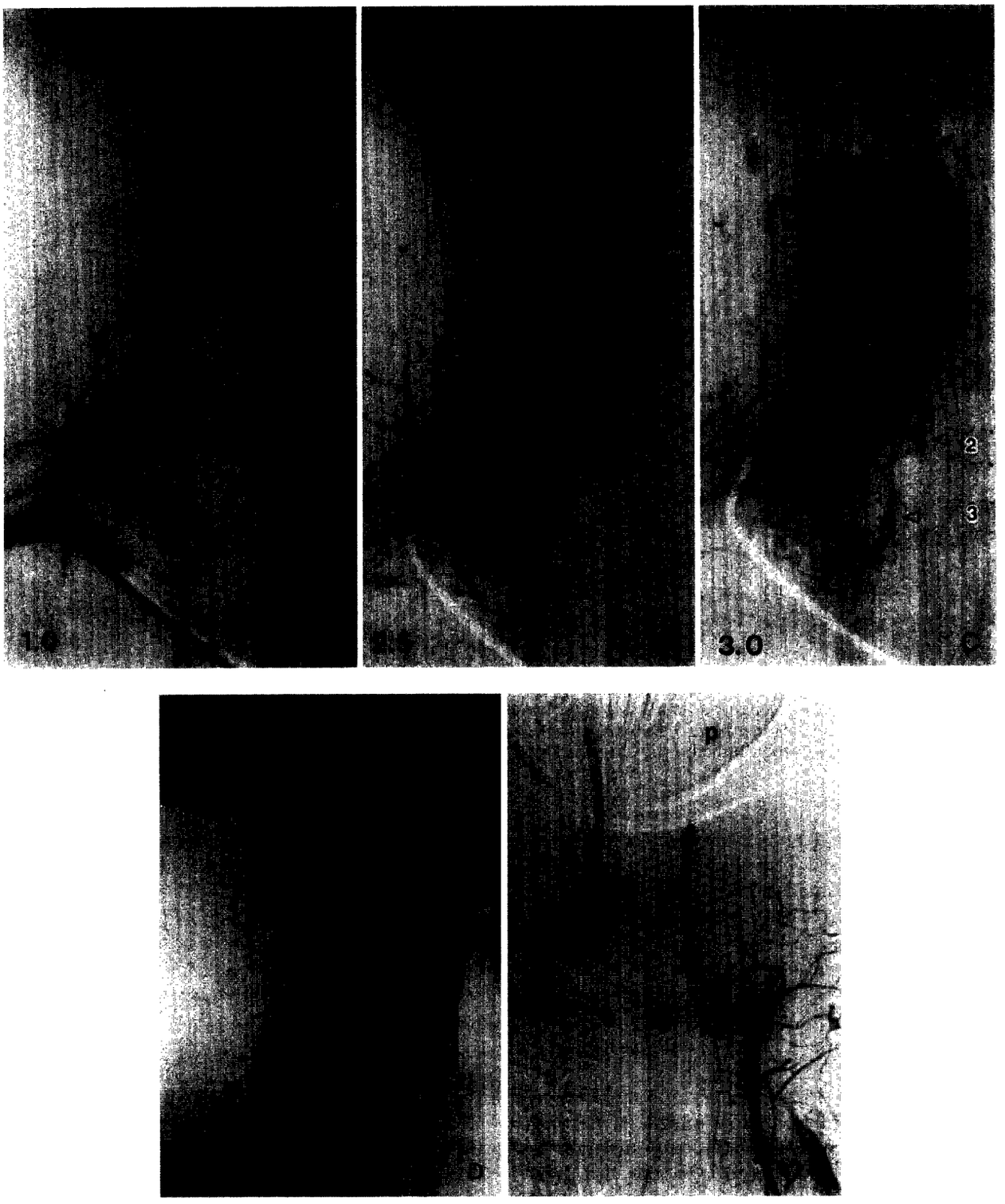
ently draining into a large, distended tortuous vein extending downward from the lake. Meticulous coagulation of these small red vessels was followed by complete occlusion of the large drainer. The venous lake was almost completely removed except for a small area tightly adherent to the pia mater. The dura-arachnoid was closed hermetically. Laminoplasty of C-4 to C-7 was performed with the excised spinal processes. The wound was closed in layers.

During the next 3 days, her symptoms were aggravated temporarily with paraplegia. Hyperbaric oxygen therapy, prostaglandin, and methylprednisolone were administered. On the 4th postoperative day, she began to move her ankle and knee joints, followed by gradual recovery. On the 58th postoperative day, she could walk unaided and was discharged. Follow-up examination in the postoperative 12th month, found neurologic abnormalities of hyperreflexia in four limbs and hypesthesia in the left C-8 dermatome. She was able to walk and run, and had resumed normal school life.

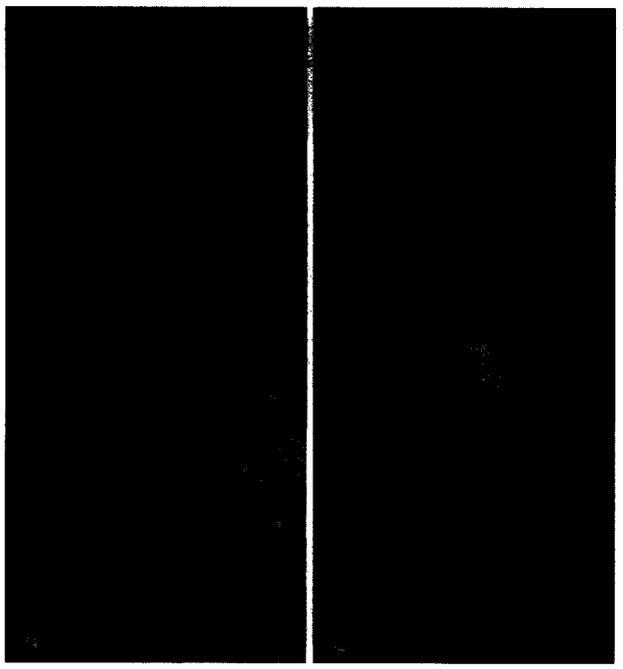
MRI performed 163 days (about 5 months) after the operation (Figure 6) revealed a re-expanded cervical cord with strand-shaped T_1 low and T_2 high-intensity areas at C-4 and 5, suggesting myelomalacia. A small area of hemosiderin deposit with an extremely low T_2 signal was noted in the cord adjacent to the myelomalacia. No further MRI follow-up or postoperative spinal arteriography were obtainable.

DISCUSSION

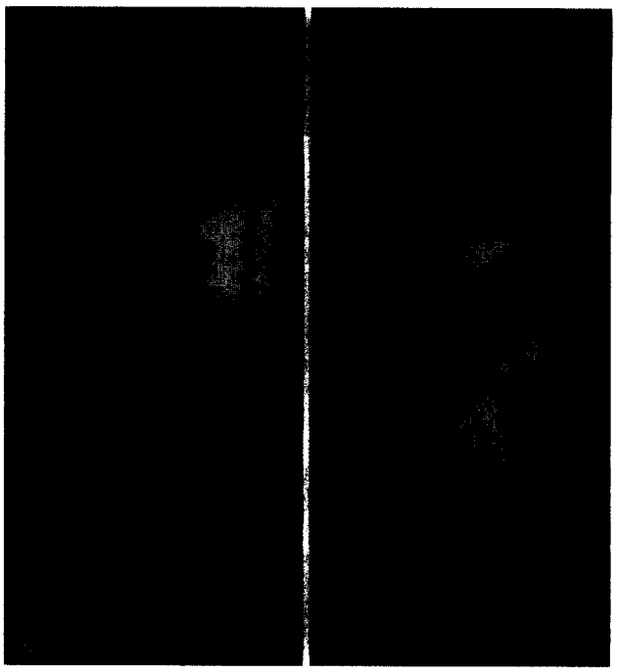
Only a few cases of intradural perimedullary arteriovenous fistula have been described since the first in 1977 by Djindjian et al [2,4,6,8–10,14,17], but in 1986 the disease was defined by Heros et al [9] as a new "Type IV" spinal arteriovenous malformation. The incidence of intradural arteriovenous fistulas is unknown, but comprised 4% (6/150) [4], 13% (11/81) [17], and 17% (35/210) [10] of large series of spinal arteriovenous malformations. "Giant" intradural arteriovenous fistula is seldom reported, although the definition of "giant" is not



4 Spinal arteriograms through the right costocervical artery injection (**A,B,C,D**) and through the highest intercostal artery injection (**E**). **A,B,C,D** are taken at 1.0, 2.5, 3.0, and 4.0 seconds after injection, respectively. The label (*cc*) in **A** indicates the costocervical artery. Note: the rapid opacification of the draining system; upward (*open arrows* in **B,C,D**) to the level of the foramen magnum, and downward to the thoracic canal (*open arrowheads* in **B,C**; (*2*) (*3*) in **C** indicate the second and the third ribs). *White arrowheads* in **A,B** indicate the site of the fistula. The label (*hi*) in **E** indicates the highest intercostal artery. **E** was taken immediately after embolization. Note the ascending branch arising from the highest intercostal artery joins the costocervical artery to form a single ascending feeder to the fistula. *P* is the platinum coil used for embolization. Neither the venous lake nor the drainers are visualized any longer.



5 Midsagittal T_1-weighted (TR/TE = 500/15 msec) spin echo (**A**), and T_2-weighted (TR/TE = 4500/103 msec) fast spin echo (**B**) MRIs demonstrating disappearance of the signal void areas, 20 days after embolization. The T_1 and T_2 high-signal intensities within the cyst reflect methohemoglobin formed in the thrombus.



6 Midsagittal T_1-weighted (TR/TE = 500/15 msec) spin echo (**A**), T_2-weighted (TR/TE = 4500/103 msec) fast spin echo (**B**) MRIs, 163 days after excision, demonstrating the re-expanded spinal cord with strange-shaped T_1 low and T_2 high intensities, suggesting myelomalacia. The extremely T_2 low signal adjacent to the myelomalacia reflects hemosiderin deposit (**B**).

clear. Halbach et al [8] simply used giant to mean "large," and Gueguen et al [6] to mean "extremely large" intradural fistulas. Fortunately, these formidable lesions are rare. In Djindjian's series of 160 spinal arteriovenous malformations [4], only one patient (0.6%) had a "pseudo-ectasic" [4] large intradural fistula. Other authors have reported a similar low incidence. In the series reported by Guenguen et al [6], 2 of 11 patients with documented intradural fistulas had a "giant" fistula.

Table 1 summarizes the 17 reported cases including our present case [2,4,6,8,10,13,14,17]. There were 10 females and 7 males aged from 3 weeks neonate [2] to 43 years [6] (mean, 21.1 years). Thirteen patients were classified as perimedullary Type III and three as Type II, with one unknown.

The clinical presentation of the majority of giant intradural arteriovenous fistulas usually involves progressive radiculomedullary symptoms; 56% (7/16) of patients presented with progressive myelopathy and 12% (2/16) with acute paraplegia or monoplegia. Guenguen et al reported that if intradural fistulas are untreated, there is a gradual progression to a disabling partial medullary syndrome, resulting in complete spinal transsection within 7 to 9 years [6]. Spinal subarachnoid hemorrhage occurred in 50% (8/16) of patients. Topographically, the fistulas are located most often at the conus medullaris or cauda equina (10/17), and less often in the cervical (4/17) and thoracic (3/17) regions.

The origin of perimedullary fistulas is unknown. In one patient, the fistula may have been related to epidural anesthesia causing trauma to a low-tethered cord and producing a single-hole arteriovenous fistula at the top of the conus. A similarly acquired fistula may have developed following excision of conus medullaris ependymoma [2]. Extremely young patients of 2 years of age [8] or neonate [2], and the association with Cobb and Rendu-Osler-Weber syndrome, suggests the possibility of congenital origins. The frequent association of Cobb, 24% (4/17), and Rendu-Osler-Weber, 11% (2/17), syndromes far exceeds the reported association of such syndromes with other spinal arteriovenous malformations (2%–3%) [5,17]. Cobb syndrome initially referred to a port-wine capillary malformation adjacent to the spinal cord malformation, but has been expanded to include metameric malformations, or arteriovenous malformation (AVM) involvement of the spinal cord, extending into the adjacent dura, vertebrae, skin, or the extremities. These strong associations suggest a link between these syndromes and giant intradural fistula [8].

1 Reported Cases of Giant Intradural Perimedullary Arteriovenous Fistula

Author(s)/ Year	Age (yr)/Sex	Presenting Symptoms	Associated Condition	Location, Type of Fistula ( )	Treatment	Follow-up Angiogram Outcome	Follow-up— Months
Djindjian et al, 1977	13/M	SAH (age 4), progressive myelopathy	—	Th4 (III)	Surg (En bloc excision with feeders)	Cure	6
Riche et al, 1982	Young/F	n. d.	Cobb	C	n. d.	n. d.	n.d.
Riche et al, 1983	34/M	SAH (age 15), progressive myelopathy		L1 (III)	Embo	Cure	5
Gueguen et al, 1987	43/M	SAH (age 21), progressive myelopathy	n. d.	L1 (III)	Embo	Cure	5
	22/M	SAH (age 19), acute left leg paresis, left Brown-Séquard syndrome	n. d.	Th11 (III)	Embo (detachable balloon)	Cure	2
Halbach et al, 1993	2/F	Progressive myelopathy	—	L (III)	Embo	Cure	112
	22/M	SAH (age 9, SAH (age 21)	—	L (III)	Embo and surg	Refused	84
	35/F	Progressive myelopathy	Cobb	L (III)	2 embo and surg	Cure	68
	27/F	Progressive myelopathy	—	L (III)	2 operations, 3 embo	Cure	58
	31/M	Progressive myelopathy	—	L (III)	2 embo and surg	Cure	45
	7/F	SAH	R-O-W	L (III)	2 embo	Cure	41
	40/F	Progressive myelopathy	—	Th (III)	2 embo and surg	Cure	28
	7/F	Acute paraplegia	R-O-W	C (II)	Embo and surg	<5% residual	5
	13/M	SAH	Cobb	L (III)	Embo and surg	Cure	4
	11/F	SAH	—	C (II)	Embo	<5% residual	3
Barrow et al, 1994	3 wks/F	Paraparesis, high-output heart failure	Cobb	T11 & L2 (III)	Embo	Cure	4
Nagashima et al, 1995	17/F	Progressive myeloradiculopathy	—	C5-6 (II)	Embo and surg	Cure	12

Abbreviations: M = male; F = female; SAH = subarachnoid hemorrhage; Th = thoracic; R-O-W = Rendu-Osler-Weber syndrome; wks = weeks; n.d. = not described; L = lumbar; C = cervical; Cobb = Cobb's syndrome; Embo = embolization; Surg = surgery.

Symptoms are frequently aggravated by certain postures or activities, such as sneezing, coughing, and straining as in the present patient or, in some patients, by pregnancy [18]. Except for the direct trauma caused by subarachnoid hemorrhage, the pathophysiologic basis for the clinical presentation remains uncertain. At least three mechanisms have been proposed:

(1) vascular steal resulting in ischemia [11,18], which is possible in our patient because the anterior spinal artery was involved in the fistula;
(2) mass effect created by an enlarged fistula or fistulas, the distended draining veins [9,11], and the venous lake [11], as occurred in the present patient with greater mass effect when thrombosis developed; and
(3) venous hypertension secondary to the anomalous shunt [18].

The latter mechanism would result in

(1) reduced arteriovenous pressure gradient,
(2) decreased intramedullary blood flow, and

(3) severe radicular pain and paralysis. In our patient, the latter symptom caused by venous hypertension became very pronounced after embolization of the feeder and led to the surgical excision.

The use of the microcatheter and platinum coils with fiber [7] resulted in complete occlusion of the fistulous site and was of great help in resecting the huge venous lake, as well as in identifying the anterior spinal artery containing the platinum coils when seen through the operating microscope. We were concerned about the effect of occluding the anterior spinal artery. Preoperative temporary embolization of the artery with negative amytal test and the absence of postembolization sequelae suggest that this artery acted merely as a short tube passing blood into the fistula, not as a normal anterior spinal artery supplying blood to the cord. Arteriography demonstrated no major collateral arterial cord supply in our patient, but a non-opacified collateral arterial cord supply must have been present. Non-opacification probably resulted from the rapid arteriovenous shunting. We felt justified in removing the thrombosed venous lake surgically. Propagation of thrombus into the extensive ascending and descending venous channels may have caused deterioration [9]. The temporary aggravation with paraplegia lasting for 3 days in our patient was probably related to the meticulous bipolar coagulation of the small pial vessels around the ventral wall of the venous lake. The left C-8 posterior rootlets may have become involved in this procedure. Retrospectively, preservation of a wider area of the ventral wall might have been preferable, so maintaining the compromised cord, root, and arterioles intact. We recommend that such a huge perimedullary arteriovenous fistula with a rapid transit time, and severe cord and root compression, should be treated with embolization followed by surgical excision.

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COMMENTARY

The authors present a rare type of intradural subpial fistula on the surface of the spinal cord with an enormous varix and venous drainage. Endoarterial embolization of a fast-flow fistula is better achieved with detachable balloon or with acrylic glue than