
Cervical Myelopathy Associated with Intracranial Dural Arteriovenous Fistula: MR Findings Before and After Treatment

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Summary: The MR findings in three patients with intracranial dural arteriovenous fistula associated with cervical myelopathy are described. The MR appearance of an enlarged cord with associated abnormal signal and enhancement is nonspecific and can simulate tumor, demyelination, and inflammation. Enlarged perimedullary vessels may not always be identifiable, but if present, should suggest the presence of an arteriovenous fistula.

Index terms: Fistula, arteriovenous; Spinal cord, myelopathy

Intracranial dural arteriovenous fistulas (DAVFs) are well-known entities that result from an abnormal communication between meningeal arteries and veins and/or venous sinuses. Intracranial DAVFs can produce a variety of clinical manifestations, including intracranial hemorrhage, ischemia/infarction, mass effect from enlarged veins, increased intracranial pressure, and cranial neuropathies. Myelopathy resulting from an intracranial DAVF is uncommon. Recognition of the magnetic resonance (MR) imaging findings is important, since many of the imaging characteristics are nonspecific. If not properly identified, a delay in diagnosis can result in progression of the myelopathy or in an unwarranted cord biopsy.

We report the MR findings in three patients in whom progressive cervical myelopathy developed as a result of intracranial DAVF. Of the 25 cases we found previously reported in the literature, the MR features were detailed in eight cases. MR findings in these eight patients ranged from normal to cord enlargement with associated signal abnormality of the cord and enlarged perimedullary vessels (1–6). We present the MR features related to this myelopathy before and after endovascular and surgical therapy and discuss the importance of identify-

ing enlarged veins adjacent to the spinal cord abnormality and of following patients after treatment.

Case Reports

Case 1

A 71-year-old man was admitted with progressive nausea and vomiting and bilateral leg weakness. MR images of the brain and cervical spine revealed abnormal hyperintensity on the long-repetition-time (TR) images in the lower medulla and upper cervical cord to the C-3 level. Enlarged perimedullary vessels were present along the lower brain stem, cervical cord, and upper thoracic cord (Fig 1A–C). Angiography revealed an intracranial DAVF of the superior petrosal sinus with perimedullary venous drainage (Fig 1D and E).

Embolization was not attempted, since the meningeal branches arising from the internal carotid artery were the predominant supply, and their safe catheterization and occlusion were not assured. Surgery was performed to obliterate the fistula. Postoperatively, the extremity weakness improved dramatically. A repeat MR examination 10 days after surgery showed improvement in brain stem signal abnormality and complete resolution of the abnormally dilated veins (Fig 1F). The patient has remained neurologically stable with improvement in leg strength over an 18-month period.

Case 2

A 47-year-old man had a 5-month history of progressive myelopathy. MR imaging showed mild cord enlargement with abnormal hyperintensity on the long-TR images. Subtle signal voids were present ventral and dorsal to the cervical cord (Fig 2A). A DAVF, which was identified medial to the occipital condyle at angiography, was supplied predominately by the right ascending pharyngeal artery (Fig 2B). The fistula was successfully treated by endovascular embolization with 0.1 mL of *N*-butyl-cyanoacrylate (Fig 2C). After embolization, mild symptomatic

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Fig 1. Case 1: 71-year-old man with DAVF of the superior petrosal sinus.

A, Sagittal fast spin-echo T2-weighted MR image (3000/96/4 [repetition time/effective echo time/excitations]) shows abnormal hyperintensity in the medulla and upper cervical cord. Abnormally enlarged veins are present ventral to the brain stem (arrow) and dorsal to the spinal cord (arrowhead).

B, Sagittal fast spin-echo T2-weighted MR image (3000/96/4) shows enlarged veins extending inferiorly to the lower thoracic region.

C, Postcontrast sagittal T1-weighted MR image (500/10/2) reveals enhancement of an enlarged venous plexus.

D, Lateral view of a selective internal carotid arteriogram shows a superior petrosal DAVF (arrow) fed by multiple branches of the meningohypophyseal trunk with an early draining vein (arrowhead).

E, Anteroposterior view of the right internal carotid arteriogram shows perimedullary venous drainage with enlarged spinal veins (arrowheads).

F, Sagittal fast spin-echo T2-weighted (3000/96/4) MR image after surgical resection shows persistent hyperintensity within the medulla, which remains mildly swollen, and disappearance of the abnormally enlarged veins.

improvement occurred with no further progression of the myelopathy. A 4-year follow-up MR study showed a normal cervical cord without enhancement or abnormally dilated veins (Fig 2D). The thoracic cord had normal signal with mild to moderate cord atrophy.

Case 3

A 58-year-old woman was known to have had an extensive DAVF of the skull base for many years. Previous treatments included attempted local resection, left external carotid artery ligation, left vertebral artery ligation, and external beam radiation. She presented with progressive cervical myelopathy. MR imaging revealed a mildly enlarged cervical cord and hyperintensity within the cord on the long-TR images. Enlarged vessels dorsal and ventral to the cord were subtle but present (Fig 3A and B).

Angiography showed an extensive DAVF of the skull base with arterial supply via collaterals to the occluded external carotid artery from the left ascending cervical, left vertebral, and left ophthalmic arteries (Fig 3C). The dural fistula was so extensive, with limited arterial access, that complete occlusion either via embolization or surgery was

not thought to be achievable. Partial embolization was performed to diminish arterial inflow, thereby decreasing venous pressure in the hope of stabilizing the patient's cervical myelopathy. Embolization was performed using polyvinyl alcohol and silk thread. After embolization, the symptoms improved transiently. Over the ensuing months, her condition worsened with increased spasticity and muscle weakness. A follow-up MR examination at 4 years showed persistent cord signal abnormality, cord swelling, and abnormal cord enhancement; however, no enlarged veins were apparent (Fig 3D). Although no enlarged perimedullary veins were identified, the patient's clinical course was compatible with a worsening fistula due to recruited collaterals or recanalization.

Discussion

Recognition of the MR findings of DAVFs is important, because most patients with symptoms of myelopathy are initially examined by MR imaging. An improper diagnosis can result in an unwarranted cord biopsy or a delay in

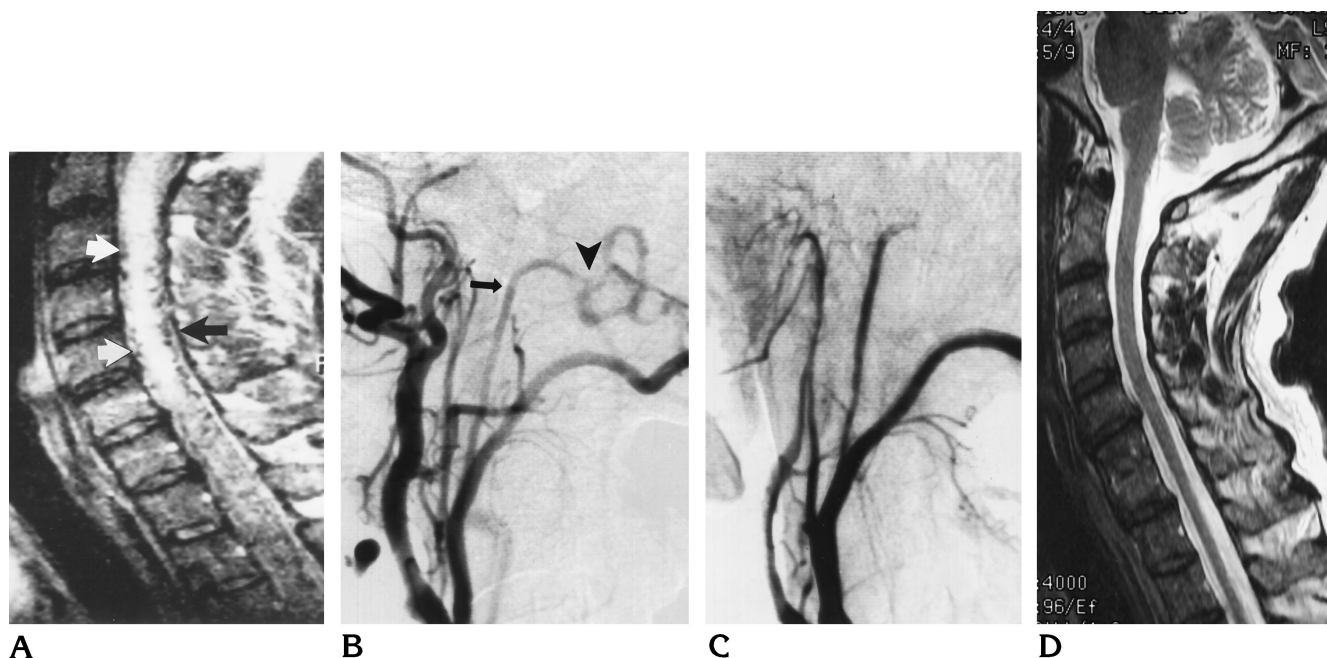


Fig 2. Case 2: 47-year-old man with DAVF medial to the occipital condyle.

A, Sagittal T2-weighted (1500/80/4) MR image shows diffuse hyperintensity within the cervical cord extending into the medulla. Enlarged veins are noted ventral and dorsal to the cord (arrows).

B, Lateral view of the left external carotid artery shows a DAVF (arrowhead) supplied by an enlarged ascending pharyngeal artery (arrow) located near the occipital condyle.

C, Postembolization external carotid arteriogram shows obliteration of the AVF. Vertebral arteriography confirmed absence of flow from other sources.

D, Sagittal fast spin-echo T2-weighted (4000/96/4) MR image at 4-year follow-up shows no abnormally enlarged vessels and normal cord signal. There is mild-to-moderate thoracic cord atrophy.

diagnosis with progression of myelopathy. These three cases are unusual because the dural fistulas were located intracranially with secondary effects on the cervical cord.

Intracranial, craniocervical, and spinal DAVFs are uncommon causes of myelopathy. Myelopathy associated with DAVFs is related to venous hypertension with subsequent edema, hypoxia, or both (7). In our three patients, the site of the fistula was remote from the level of the spinal cord dysfunction.

Wrobel et al (6) described three patients who had cranial DAVFs with associated myelopathy that were documented by angiography. In all three cases, the DAVFs were primarily supplied by the carotid circulation. Venous drainage communicated to the posterior spinal veins via the petrosal vein. These cases support the theory that neurologic impairment is related to venous hypertension and not arterial insufficiency.

Unlike parenchymal arteriovenous malformations, the abnormal arteriovenous communication in DAVFs is within the dura or the wall of the dural sinus. The nidus is usually small and

often not identifiable on routine spin-echo MR images. MR findings of intracranial DAVFs are often subtle and include enlarged venous structures, occluded venous sinuses, associated parenchymal edema, infarction, and hemorrhage (8). These findings usually relate to the diversion of venous flow from the sinuses to the cerebral veins. Flow-related enhancement within and enlargement of the venous sinuses may be present on three-dimensional time-of-flight images (9).

In our three cases of intracranial DAVF with associated cervical myelopathy, the MR findings included cervical cord enlargement and abnormal hyperintensity on the long-TR images within the medulla and cervical cord. Serpiginous areas of signal void located ventral and dorsal to the cord representing enlarged perimedullary veins were present in all cases. Contrast material was administered intravenously in only one case and produced enhancement of the enlarged venous plexus. On the basis of findings in spinal DAVFs, variable parenchymal and vascular enhancement would not be unex-

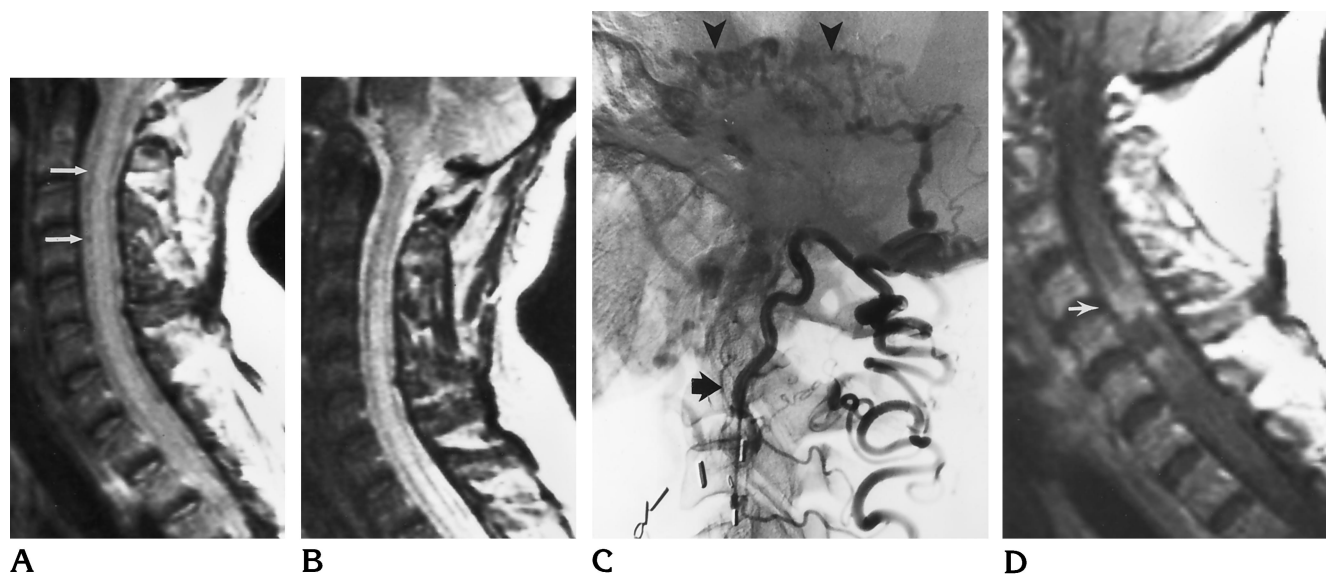


Fig 3. Case 3: 58-year-old woman with extensive DAVF of the skull base.

A, Sagittal proton density-weighted (2000/45/4) MR image shows subtly enlarged veins ventral to the cervical cord (*arrows*).

B, Sagittal T2-weighted (2000/90/4) MR image shows hyperintense signal throughout the cervical cord extending into the medulla.

C, Lateral view shows ascending cervical artery reconstituting left occipital artery (*arrow*), which supplies an extensive skull-base dural fistula (*arrowheads*). The proximal occipital artery had previously been occluded surgically. After embolization of the fistula, decreased but persistent flow to the DAVF was noted.

D, Sagittal T1-weighted (500/10/2) MR image at 4-year follow up shows prominent enhancement within a swollen cervical cord (*arrow*). Persistent hyperintensity was noted within the cord on the T2-weighted image (not shown).

pected (10). The MR findings of an enlarged cord with abnormal signal are nonspecific and can easily be mistaken for tumor, demyelination, or myelitis. The identification of enlarged veins should suggest the presence of a DAVF, which can be confirmed with angiography.

Similar changes—including cord signal abnormality, cord enlargement, and enlarged perimedullary vessels—are seen with spinal DAVFs (11, 12). Spinal DAVFs may produce changes at levels above or below the fistula itself, owing to transmission of venous hypertension via the coronal venous plexus. However, we have not found signal abnormalities of spinal DAVFs reported to extend into the medulla, as in our three cases. The findings of intracranial DAVFs on routine spin-echo MR sequences are subtle and may not be apparent. Therefore, negative findings at spinal angiography performed to search for a spinal DAVF should be followed by cerebral angiography to exclude a cranial DAVF with spinal venous drainage. In addition, sacral DAVFs can occur with ascending venous engorgement and venous hypertension leading to thoracic myelopathy. Therefore, if both the spinal and cerebral arteriograms are negative for the presence of a DAVF, a flush aortogram to exclude a sacral DAVF is indicated.

The abnormal signal within the cord and associated cord enlargement is thought to represent edema from venous hypertension. Associated cord ischemia with demyelination and/or venous infarction may occur, which may account for the persistent neurologic deficit following successful treatment of the fistula. The serpiginous areas of signal void around the cord represent enlarged veins. Demonstrated caudal flow via intracranial-to-spinal venous communications explains the transmission of venous hypertension to the coronal venous plexus of the upper spine. On the basis of MR findings in cases of spinal DAVFs, areas of signal void identifiable on routine spin-echo images would not be expected in all cases of intracranial DAVFs. Bowen et al (13) identified enlarged vessels on routine MR images in five of eight cases of spinal DAVFs. Gilbertson et al (14) identified flow voids on long-TR images in 45% of 44 cases of spinal DAVFs. MR angiography may be beneficial, especially in cases of suspected DAVF in which there are no identifiably enlarged vessels on routine spin-echo images. Bowen et al (13) identified abnormal vessels in all eight of their cases at MR angiography after contrast administration.

On the basis of our findings and on reports in

the literature, the radiologic evaluation of suspected spinal DAVFs should include a routine contrast-enhanced MR examination of the spine. If abnormal signal is present within the cord on long-TR images, and no abnormal veins are identified on the spin-echo images, then MR angiography and myelography are recommended. Angiography is required to confirm the presence of and to characterize the fistulous site.

Analogous to the treatment of spinal DAVFs, the goal of therapy is complete occlusion of the intracranial dural fistula, thereby stabilizing or reversing neurologic symptoms (3, 15). Improvement of the myelopathy varies after treatment depending on the duration of the fistula and the degree of permanent cord damage; however, stabilization of signs and symptoms is usually achieved. Complete reversal of clinical deficits was not achieved in any of our patients despite the apparent cure in cases 1 and 2. Multiple previous surgical procedures led to limited arterial access in patient 3 and prohibited complete obliteration, allowing for subsequent deterioration.

In conclusion, we documented the MR findings in three patients who had intracranial DAVFs with associated cervical myelopathy. Findings included abnormal cervical cord signal extending into the medulla, mild cord enlargement, and the identification of enlarged perimedullary veins. On the basis of findings in cases of spinal DAVFs, abnormally enlarged perimedullary vessels would not be expected in all cases. In such cases, a high degree of clinical suspicion in association with the nonspecific findings of cord swelling and edema is required to suggest the diagnosis of a DAVF. These cases emphasize that DAVFs may be remote from the level of myelopathy, which is important when performing angiography in suspected cases of spinal DAVFs. MR imaging is useful in following up patients after treatment to evaluate cord signal abnormality and dilated veins.

References

1. Bret P, Salzman M, Bascoulegue Y, et al. Dural arteriovenous fistula of the posterior fossa draining into the spinal medullary veins: an unusual cause of myelopathy. *Neurosurgery* 1994;35:965-968
2. Cognard C, Gobin Y, Pierot L, et al. Cerebral dural arteriovenous fistulas: clinical and angiographic correlation with a revised classification of venous drainage. *Radiology* 1995;194:671-680
3. Gobin P, Rogopoulos A, Armond A, et al. Endovascular treatment of intracranial dural arteriovenous fistulas with spinal perimedullary venous drainage. *J Neurosurg* 1992;77:718-723
4. Partington M, Rufenacht D, Marsh W, et al. Cranial and sacral dural arteriovenous fistulas as a cause of myelopathy. *J Neurosurg* 1992;76:615-622
5. Versari P, D'Aliberti G, Talamonti G, et al. Progressive myelopathy caused by intracranial dural arteriovenous fistula: report of two cases and review of the literature. *Neurosurgery* 1993;33:914-919
6. Wrobel C, Oldfield E, DiChiro G, et al. Myelopathy due to intracranial dural arteriovenous fistulas draining intrathecally into spinal medullary veins. *J Neurosurg* 1988;69:934-939
7. Aminoff M, Barnard R, Logue V, et al. The pathophysiology of spinal vascular malformations. *J Neurol Sci* 1974;23:255-263
8. DeMarco J, Dillion W, Halbach V, et al. Dural arteriovenous fistulas: evaluation with MR imaging. *Radiology* 1990;175:193-199
9. Chen J, Tsuruda J, Halbach V, et al. Suspected dural arteriovenous fistula: results with screening MR angiography in seven patients. *Radiology* 1992;183:265-271
10. Terwey B, Becker H, Thron A, et al. Gadolinium-DTPA enhanced MR imaging of spinal dural arteriovenous fistulas. *J Comput Assist Tomogr* 1989;13:30-37
11. Masaryk T, Ross J, Modic M, et al. Radiculomeningeal vascular malformation of the spine: MR imaging. *Radiology* 1987;164:845-849
12. Minami S, Tadashi S, Kazumasa N, et al. Spinal arteriovenous malformation: MR imaging. *Radiology* 1988;169:109-115
13. Bowen B, Fraser K, Kochan J, et al. Spinal dural arteriovenous fistulas: evaluation with MR angiography. *AJNR Am J Neuroradiol* 1995;16:2029-2043
14. Gilbertson J, Miller G, Goldman M, et al. Spinal dural arteriovenous fistulas: MR and myelographic findings. *AJNR Am J Neuroradiol* 1995;16:2049-2057
15. Oldfield E, DiChiro G, Quindlen E, et al. Successful treatment of a group of spinal cord arteriovenous malformations by interruption of the dural fistula. *J Neurosurg* 1993;59:1019-1030