Spatial distribution and postoperative changes in intramedullary lesions caused by spinal dural arteriovenous fistulas of the thoracic cord

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ABSTRACT

Objective: Spinal dural arteriovenous fistulas (AVFs) represent a rare but important cause of myelopathy brought about by venous hypertension due to shunt formation between the dural artery and medullary vein. Magnetic resonance imaging (MRI) findings include intramedullary hyperintensity on T2-weighted images, as well as cord enlargement due to venous hypertension. Postoperative resolution patterns of hyperintensity suggest a pathophysiology of spinal dural AVF.

Clinical presentation and results: We report two cases of thoracic spinal dural AVF with a single feeding artery, which were treated by surgical intervention. MRI findings were reviewed before and after surgery. Before surgery, hyperintensity on T2-weighted images spread from above the feeding artery to the conus, and was present in the central cord on axial images. After correction of the arteriovenous shunt, the region of hyperintensity began to resolve from the cranial side followed by the caudal side.

Conclusion: Hyperintensity on T2-weighted images of the cord was largely reversible, indicating that edema of the cord was caused by elevated venous pressure. The hyperintensity on T2-weighted images decreased initially from the cranial side and then from the caudal side. This transmission and resolution of increased venous pressure appeared to depend on the presence of obstruction to the venous outflow system of the spinal cord.

Key word: spine, dural arteriovenous fistula, magnetic resonance imaging, vein

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Introduction

Spinal dural arteriovenous fistulas (AVFs) represent a rare but important cause of myelopathy [6, 12]. The pathophysiology of AVF is characterized by venous hypertension due to shunt formation between the dural artery and medullary vein [3]. Magnetic resonance imaging (MRI) of dural AVFs show a dilated coronary venous plexus and intramedullary signal changes, namely hyperintensity.
on T2-weighted images and cord enlargement [2, 6, 8]. These are thought to be reversible processes and usually disappear after correction of the shunt. The cord enlargement and/or hyperintensity on T2-weighted images involves the conus with variable cephalad extension [13]. However, the distribution and the resolution of hyperintensity on T2-weighted images have not been well described.

In this report we document the MRI findings in two patients with single feeder spinal AVF of the thoracic cord, before and after surgery. We also make special reference to hyperintensity on T2-weighted images, and discuss the pathophysiology of spinal AVF.

Clinical Material and Methods

Two cases of thoracic spinal AVF with a single feeding artery were evaluated. Both patients had angiographically proven spinal dural AVF with a single feeder. MRI examinations were performed before and after surgery, and the distribution of the intramedullary hyperintensity was evaluated. MRI was performed using a 1.5-Tesla superconducting apparatus. T2-weighted sagittal and axial spin-echo images (repetition time/echo time/excitations: 4000/112/3) were obtained.
Case Reports

< Case 1 >

A 74-year-old male patient noticed numbness in his right leg 2 years before admission. The numbness gradually became worse, and weakness and numbness was evident in both legs after 1 year. He began to use a cane, and later a wheelchair. He was referred to the Department of Neurology of our hospital for evaluation. Imaging work-ups revealed a spinal AVF, so he was then referred to our department. The patient had a history of bladder cancer, and had undergone radical cystectomy and reconstruction with an ileal conduit.

On admission, paraparesis was more evident on the left side than on the right, with hyphesia below level Th 12. Patellar and achilles tendon reflexes were increased on both sides and the patient complained of severe intermittent pain in his perianal area. MRI of the thoracic cord revealed flow void signals dorsally and an area of intramedullary T2 elongation from Th 4 to the conus (Fig. 1a). The central cord area showed hyperintensity on T2-weighted images (Fig. 1e). Spinal angiography showed a dural AVF in the tributary of the left 5th intercostal artery (Fig. 2). A dilated coronal venous plexus was noticed from Th 3 through Th 8.

The patient underwent laminectomy from Th 4 to Th 6 to correct the dural AVF. Paraparesis improved after surgery, allowing him to walk with a cane. Correction of the left Th 5 fistula was confirmed by spinal angiography.

MRI scans were repeated 3 weeks, 2 months and 3 months after surgery. Three weeks after surgery, intramedullary T2 elongation was present from Th 6 to the conus (Fig. 1b). Two months after surgery, an area of T2 elongation was recognized from Th 6 to Th 10 (Fig. 1c). Three months after surgery, the change was limited to the area from Th 6 to Th 8 (Fig. 1d).

< Case 2 >

A 74-year-old male patient began to notice dragging of his left leg 2 years prior to admission. About a year later, he noticed weakness in his right leg. These symptoms worsened occasionally on exercise but improved after resting. He was referred to our department for treatment. On admission, he could stand but not walk, and had paraparesis graded as 3/5. His patellar and achilles tendon reflexes were normal. He had hyphesia to touch and pain below the dermatome of L5. MRI revealed flow void signals located dorsally and an area of intramedullary T2 elongation from Th 6 to the conus (Fig. 3a). Axial images revealed that the hyperintensity on T2-weighted images was located in the central cord area (Fig. 3d). Spinal angiography revealed an AVF in the tributary of the right 8th intercostal artery (Fig. 4). A dilated coronal venous plexus was noticed from Th 6 to Th 10.

The patient underwent laminectomy from Th 7 to Th 9 to correct the AVF. Sensory disturbances improved after surgery, allowing him to walk with a cane. MRI scans were repeated 3 weeks and 3 months after surgery. Three weeks after surgery, the intramedullary T2 elongation was limited to the area.
Fig. 3. Left Th 8 dural AVF (patient 2). (a) Preoperative, mid-sagittal, T2-weighted image demonstrates an area of hyperintensity in the central cord from Th 6 to the conus. Note the swelling of the cord and flow voids of the dorsal cord. (b) Postoperative (3 weeks), T2-weighted image reveals an area of hyperintensity from Th 7 to T 11. (c) Postoperative (3 months), T2-weighted image shows an area of hyperintensity from Th 8 to Th 10. (d) Preoperative, T-2 weighted axial image at the level of Th 9. The area of hyperintensity is present in the central cord region.

from Th 7 to Th 11 (Fig. 3b). Three 3 months after surgery, the original findings were limited to the area from Th 9 to Th 11 (Fig. 3c).

Results

Both patients showed hyperintensity on preoperative T2-weighted images extending from above the feeding artery down to the conus. The cephalad margins of T2 elongation were located at approximately the same level as the upper end of the dilated coronal venous plexus, as observed by spinal angiography. In both patients, T2 elongation was located in the central cord region.

After correction of the arteriovenous shunt, the area of T2 elongation began to resolve from the cranial side followed by the caudal side. Three months after surgery, it was limited to approximately three segments surrounding the feeder. The patients’ clinical symptoms improved despite residual signs of hyperreflexia.

Discussion

The hyperintensity seen in the thoracic cord to the conus on T2-weighted images decreased after correction of the shunt. Therefore, the hyperintensity in the cord was largely reversible, indicating that edema of the cord was caused by elevated venous pressure [6]. The longitudinal distribution of hyperintensity extended from a few levels above the feeder and caudally to the conus. Bowen et al. reported that the hyperintensity on T2-weighted images usually involved about five levels, not including the conus [2]. In contrast, Willinsky et al noticed hyperintensity on T2-weighted images
involved the conus in all cases [14]. The upper limit of hyperintensity was consistent with the dilated coronal venous plexus seen on spinal angiography, whereas the lower limit was more caudal to the angiographic abnormality. As the hyperintensity in the cord persisted for more than 3 months after surgery, irreversible processes such as necrosis and gliosis were probably involved [12].

Dural arteriovenous malformation is now considered to be an acquired disease process following trauma and thrombosis [1, 9]. The artery splits into daughter vessels in the dura, which invariably empty into a medullary vein without an intervening capillary process. If drainage of the anterior median spinal vein is obstructed, however, direct fistulae can be opened because of the lack of an anti-reflux valve [9]. The nidus of the fistula is usually present within the intervertebral foramen and adjacent lateral aspect of the spinal canal. Medullary veins drain the fistula in a retrograde manner into the dilated, tortuous coronal plexus of the dorsal pial surface of the spinal cord [3]. Although dilation of veins occurred on the dorsal surface of the spinal cord, the area of hyperintensity seen on T2-weighted images presented in the central cord, which is the main site of edema [5].

Gililian reported that the intrinsic spinal cord veins consist of two groups: a radial group and an anterior median group [4]. The radial group collects blood from the periphery of the gray matter and the white matter joining the coronal venous plexus. Dural AVFs occur between the dural artery and medullary vein, which drains the fistula in a retrograde manner into the coronal plexus of the dorsal pial surface of the spinal cord [3]. The posterior longitudinal venous systems are less numerous in the thoracic region and drain into an anterior median group. The anterior median group collects the venous blood from the central cord via the central vein and empties into the anterior median spinal vein. The anterior median spinal vein is well developed and connected in part to the medullary veins, which accompany the nerve roots. Among the roots of the cauda equina is a very large vein called the great anterior medullary vein.

The increased venous pressure may have been transmitted to the central vein which empties into the anterior median spinal vein. The cord edema may have been due to increased venous pressure, propagated caudally to the conus. If the great anterior medullary vein is patent, elevated venous pressure would be reduced and edema would not be seen extending into the conus.

The elevated venous pressure was reduced after the shunt had been corrected, as the arterial blood flow had been removed and venous congestion began to improve. Consequently, hyperintensity on T2-weighted images decreased, initially from the cranial side and then from the caudal side. The cephalad extension of the edema subsided soon after surgery. Willinsky et al. described that hyperintensity did not extend above Th 4 in any of their patients, and this was consistent with our results [14]. This suggests that venous hypertension subsided with patent radiculomeningeal veins, which usually drain into the superior vena cava via the azygos vein. The caudal
extension of the edema took more time to regress due to incomplete venous drainage and partly due to gravity [6]. The drainage system does not function well immediately because the main venous outflow system may still be obstructed and considerable endothelial injury may have occurred to the venous system. Besides the obstruction of venous outflow systems, vasoparalysis of the vascular bed and the “perfusion breakthrough” mechanism may also contribute to the delay in resolution of the edema [11].

Although not performed in this study, an investigation of the venous system, especially the great anterior medullary vein, would be helpful for evaluating patients with spinal dural AVF with conus edema [10].

References

Reviewer's comment:
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Akimura et al. present us an important diagnostic aid regarding MRI findings in spinal arteriovenous fistula (AVF), particularly the intramedullary changes shown by the T2-weighted images after obliteration of the arteriovenous shunt by surgical management.

There are variable reports regarding the longitudinal distribution of hyperintensity in the spinal cord extending from a few levels above the feeder down to the conus medullaris. Many authors mentioned most rostral level of the areas with increased signal moved in a caudal direction. The authors suggested a spinal cord edema may have been due to increased venous pressure, propagated caudally to the conus. Kohno et al (Reference 7) suspected the gravity influenced the congestive edema, resulting in a high incidence of involvement of the conus medullaris. However, the reason why the conus medullaris is usually involved in the lesion has still not been resolved. Further study will be necessary to investigate the pathophysiology of the conus lesion in the spinal dural AVF.

These MRI findings of the spinal dural AVF associated with conus abnormalities are extremely important to make an early detection of spinal dural AVF and an evaluation of the clinical outcome.

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The authors described a course of intramedullary MRI signal alterations in two cases of thoracic dural arteriovenous fistulas. Following surgical extirpation, resolution from the cranial aspect of the fistula followed by the caudal aspect is reported, which is certainly noteworthy in consideration of the mechanism of the T2 elongation. Pathophysiology of the circulatory disturbance under the condition of AV fistula is yet to be elucidated, and readers are introduced to several theories for such mechanisms.