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Spinal cord edema: unusual magnetic resonance imaging findings in cervical spondylosis

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Object. Spinal cord edema is a rare radiological finding in chronic degenerative disorders of the spine. Between 1997 and 2001, the authors treated six patients with cervical spondylotic myelopathy in whom postoperative spinal cord edema was demonstrated. The authors describe the radiological and clinical features of this unusual condition.

Methods. The six patients were all men, and ranged in age from 44 to 72 years. All patients presented with mild quadriparesis and underwent laminoplasty or anterior fusion. Preoperative magnetic resonance (MR) imaging revealed marked spinal cord compression with intramedullary hyperintensity on T_2-weighted sequences and spinal cord enhancement at the compression level after administration of Gd.

After surgery, spinal cord edema was observed in all patients; the spinal cord appeared swollen on the postoperative MR images. Preoperative and postoperative Gd-enhanced MR imaging demonstrated clear enhancement of the white matter at the compressed segment. Neurologically, five of six patients experienced good improvement of symptoms; however, the spinal cord edema as documented on follow-up MR imaging persisted for several months after surgery.

Conclusions. The radiological characterization of spinal cord edema was based on the reversible white matter lesion most likely caused by disturbed local venous circulation induced by chronic spinal cord compression. Such unusual MR findings in cervical spondylotic myelopathy should be differentiated from intramedullary spinal cord tumors, demyelinating disorders, or inflammatory processes.

KEY WORDS • cervical spondylosis • spinal cord edema • magnetic resonance imaging

INTRAMEDULLARY hyperintensity on T_2-weighted MR imaging of the cervical spinal cord is often observed in patients with chronic spinal canal stenotic lesions such as cervical spondylosis or OPLL.[1,2,4,13,16,17,26] Such intramedullary hyperintensity has been presumed to be microcystic degeneration (myelomalacia), gliosis, demyelination, or edema in the spinal cord resulting from a long-standing compressive effect.[26] Although several pathological conditions have been proposed as the origins of the abnormal signal, it is likely that microcystic degeneration of the gray matter and associated white matter degeneration underlie most of the intramedullary signal changes in cervical spondylosis.[1,8,9,11,19,20] Spinal cord edema is a common finding in acute spinal cord injury, neoplastic, or inflammatory lesions in the spinal canal; however, it is rare in chronic degenerative disorders of the spinal column. In this report, we describe six patients with cervical spondylosis and spinal cord edema demonstrated by MR imaging. None of these patients had a history of acute spinal trauma. Clinical and radiological characteristics and possible pathogenesis of this unusual MR finding are described.

Abbreviations used in this paper: CSF = cerebrospinal fluid; CSM = cervical spondylotic myelopathy; MR = magnetic resonance; OPLL = ossification of the posterior longitudinal ligament.

Clinical Material and Methods

Between 1997 and 2001, 190 patients with cervical spondylosis (148 cases) or OPLL (42 cases) underwent surgery at our hospital. In six patients (3%) spinal cord edema was revealed on pre- and postoperative MR imaging examinations. Data obtained in these six patients were retrospectively analyzed to characterize clinical and radiological features of this unusual presentation of cervical spondylosis. All patients were men ranging in age from 44 to 72 years (mean 54 years). On admission, all patients exhibited upper-extremity motor and sensory disturbance with long tract signs or symptoms such as abnormal plantar reflexes (five cases) or motor weakness of legs (five cases). Initial symptoms were mainly upper-extremity numbness (five cases). Histories of acute trauma or episodes suggesting the inflammatory process were absent in all patients. Duration from onset of clinical symptoms to surgery ranged from 2 to 18 months (Table 1).

TABLE 1

*Summary of clinical data obtained in six patients with cervical spondylosis**

Case No.	Age (yrs), Sex	Initial Symptom	Neurological Findings on Admission	Duration From Onset to Op
1	62, M	bilat UE numbness	mild quadriparesis (UE: 4/5, LE: 4–5/5); bilat Babinski sign (+); SD of bilat UE & LE	18 mos
2	44, M	gait disturbance	mild quadriparesis (UE: 4/5, LE: 4–5/5); bilat Babinski sign (+); SD of bilat UE & LE	4 mos
3	72, M	rt UE pain & motor weakness	mod quadriparesis (UE: 4/5, LE: 4/5); bilat Babinski sign (+); SD of bilat UE & LE	14 mos
4	51, M	lt UE numbness	mild hemiparesis (lt UE: 4–5/5, lt LE: 4–5/5); dysesthesia of lt UE & LE	7 mos
5	50, M	rt UE numbness	motor weakness of UE (4–5/5); rt Chadok sign (+); SD of rt UE & LE	2 mos
6	45, M	rt UE & LE numbness	mild quadriparesis (UE: 4–5/5, rt LE: 4/5); bilat Babinski sign (+); dysesthesia of rt UE	3 mos

* LE = lower-extremity; SD = sensory disturbance; UE = upper-extremity; + = positive.

Results

Preoperative Radiological Findings

In all patients plain radiography demonstrated spondylotic changes of the cervical spine such as osseous spur formations at the middle to lower cervical levels. Preoperative MR imaging examinations revealed marked spinal cord compression at single disc levels (C5–6 in five patients and C7–T1 in one patient), with extensive intramedullary hyperintensity on T_2-weighted images, although spinal canal stenosis and mild spinal cord compression were also noted at other levels in five of six patients (Table 2). At these levels of marked compression, the spinal cord was pinched by the protruded discs and osseous spurs anteriorly, and by the yellow ligaments posteriorly. The intramedullary signal change was seen at the intervertebral disc level and the adjacent area immediately below the disc level. The intramedullary hyperintensity extended for one or two vertebral body levels from the compressed disc level in three patients (Cases 1, 3, and 4). Careful evaluation of MR images revealed that the spinal cord immediately below the disc level of marked compression was slightly or moderately enlarged in all patients. No abnormal signal intensity in the spinal cord was demonstrated on T_1-weighted MR images. Four patients underwent preoperative Gd–diethylenetriamine pentaacetic acid MR imaging, which demonstrated enhancement of the spinal cord at the marked compression level.

Surgery and Postoperative Course

Four patients (Cases 1, 3, 4, and 6) underwent posterior decompressive laminoplasty, whereas anterior decompression and fusion were performed in two patients (Cases 2 and 5). Postoperative MR imaging examinations performed at 1 to 6 weeks (1 week, four patients; 4 weeks, one patient; 6 weeks, one patient) revealed that the previously compressed spinal cord appeared swollen with increased intramedullary hyperintensity in all patients. There were no intraoperative injuries. Clinically, four patients (Cases 2–5) exhibited improvement in upper- and lower-extremity motor weakness. In one patient (Case 6) upper-extremity dysesthesia resolved. There was no change of clinical symptoms in one patient (Case 1), although his Babinski sign became negative. Postoperative Gd-enhanced MR imaging showed enhancement of the spinal cord at the compressed segment in all patients. Axial images of Gd-enhanced MR imaging revealed that the intramedullary enhancement was present in the white matter.

Follow-Up Results

Follow-up MR imaging periods ranged from 3 to 46

TABLE 2

*Summary of radiological findings**

Case No.	Level of Compression	Preop MRI		Op (levels)	Follow-Up MRI (interval from op)
		IMH	Gd Enhancement		
1	C4–5 & C5–6	C5–T1	C5–6	laminoplasty (C4–7)	atrophic cord & IMH at C5–6 (82 mos)
2	C5–6	C5–6	C5–6	ant fusion (C5–6)	cord swelling & Gd at C5–6 (3 mos)
3	C6–7 & C7–T1	C7–T1	C7–T1	laminoplasty (C3–7)	mild cord swelling & Gd at C7–T1 (11 mos)
4	C5–6	C5–6	C5–6	laminoplasty (C3–7)	cord swelling improved; IMH at C5–6 (19 mos)
5	C4–5 & C5–6	C5–6	no exam†	ant fusion (C4–5 & C5–6)	cord swelling improved; IMH at C5–6 (22 mos)
6	C3–4 & C6–7	C5–6	no exam†	laminoplasty (C3–7)	atrophic cord & IMH at C5–6 (27 mos)

* ant = anterior; IMH = intramedullary hyperintensity on T_2-weighted MR imaging.

† No preoperative Gd-enhanced MR imaging examinations were performed. Postoperative Gd-enhanced imaging, however, revealed intramedullary enhancement at the level of intramedullary hyperintensity.

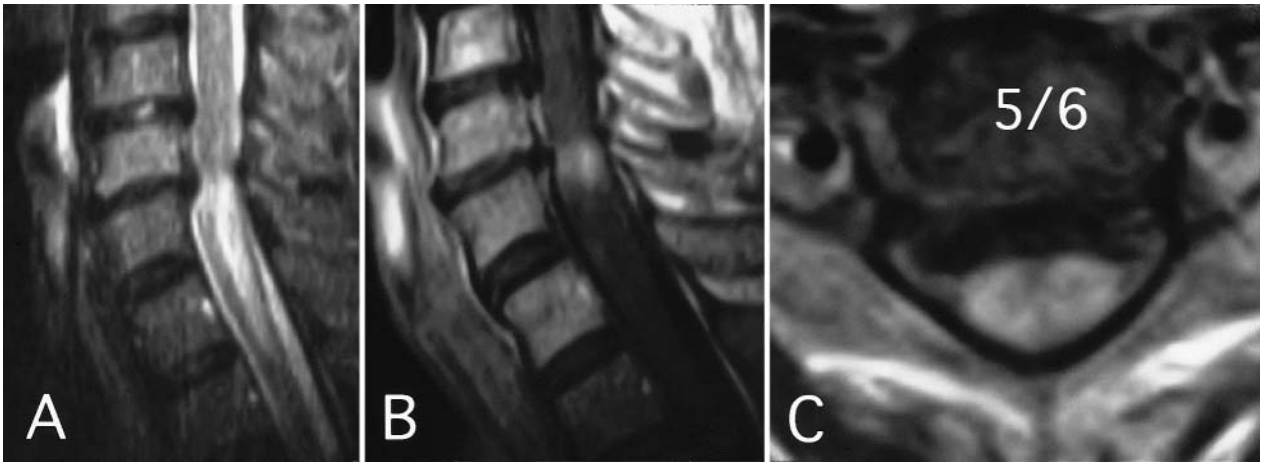

FIG. 1. Case 1. Preoperative MR images. A: Sagittal T_2-weighted image revealing a narrowed spinal canal from C4–5 to C5–6. The spinal cord is markedly compressed at C5–6. Intramedullary hyperintensity extends from C5–6 to T-1. B: Sagittal image of T_1-weighted Gd-enhanced image demonstrating intramedullary enhancement of the spinal cord at C5–6. C: Axial T_1-weighted Gd-enhanced image obtained at C5–6 revealing that the white matter of the spinal cord is mainly enhanced.

months (mean 21 months). Spinal cord swelling was present at least 3 months after surgery. In four patients with longer follow-up periods (19–46 months), the spinal cord swelling gradually subsided and returned to the normal size (Cases 4 and 5) or resulted in mild atrophy (Cases 1 and 6) at the previously compressed segment. Intramedullary hyperintensity visualized on follow-up T_2-weighted MR images was limited to the small segment at the disc level in these patients.

Illustrative Cases

Case 1

Presentation and Examination. This 62-year-old man first presented with numbness of both hands in February of 1996. Bilateral upper- and lower-extremity motor weakness gradually developed. He was admitted to our hospital on July 4, 1997. On admission, we observed motor weakness of the upper (below the triceps muscles) and lower extremities bilaterally as well as an unsteady gait. Babinski reflex was positive bilaterally. Superficial sensations of the upper extremities were decreased moderately below the elbow level. Radiographic studies demonstrated spondylotic changes of the cervical spine at C4–5 and C5–6. Magnetic resonance imaging revealed marked C5–6 cord compression. A striking spindle-shaped intramedullary hyperintensity was demonstrated from C5–6 to T-1 on T_2-weighted MR images (Fig. 1A). Gadolinium-enhanced MR images demonstrated spinal cord enhancement at C5–6 level (Fig. 1B). Axial Gd-enhanced MR imaging revealed that the bilateral white columns were mainly enhanced (Fig. 1C). Angiography revealed no vascular abnormality bilaterally.

Operation and Postoperative Course. The patient underwent expansive C4–7 laminoplasty on August 8, 1997. There was no improvement in clinical symptoms in the early postoperative period. Four weeks after surgery MR imaging demonstrated intramedullary hyperintensity extending to the C-4 level (Fig. 2A). The spinal cord swelling was more prominent compared with its preoperative appearance. There was no Gd-related change in enhancement of the spinal cord at C5–6 (Fig. 2B). The patient exhibited slight improvement in upper- and lower-extremity motor weakness during the 1-year postoperative course. Follow-up MR images revealed a gradual decrease in the intramedullary hyperintensity for a duration of 9 months. At 46 months postoperatively, T_2-weighted MR imaging revealed atrophic change with small intramedullary hyperintensity at C5–6 (Fig. 2C).

Case 2

Presentation and Examination. This 44-year-old man first presented with motor weakness in his legs in July 2001. Bilateral upper-extremity numbness and motor weakness gradually developed. He was admitted to our hospital on November 10, 2001. On admission, we observed mild motor weakness of the upper and lower extremities with bilateral extensor plantar responses. Superficial sensation was mildly decreased at both upper extremities. Radiographic studies revealed spondylotic changes of the cervical spine at C5–6. Magnetic resonance imaging revealed spinal cord compression with intramedullary hyperintensity at C5–6 on T_2-weighted sequences (Fig. 3A). The compressed spinal cord was enhanced on MR imaging after administration of Gd (Fig. 3B).

Operation and Postoperative Course. The patient underwent anterior discectomy and osteophysectomy at C5–6 as well as subsequent anterior interbody fusion involving placement of a threaded titanium cage packed with autologous cancellous bone harvested from the iliac crest on November 22, 2001. Postoperatively, good improvements of upper- and lower-extremity motor weakness as well as upper-extremity sensory disturbance occurred. One month after surgery MR imaging revealed that the decompressed spinal cord appeared swollen and that the intramedullary hyperintensity was increased on axial T_2-weighted se-

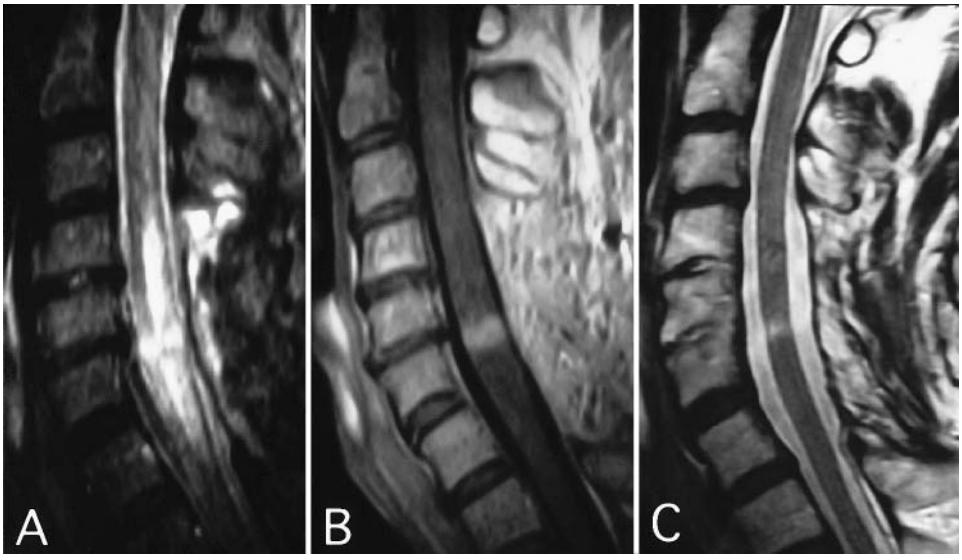

FIG. 2. Case 1. Postoperative MR images. A: Sagittal T_2-weighted image obtained 4 weeks after the laminoplasty. The intramedullary hyperintensity extends to C-4. The spinal cord appears swollen. B: Sagittal T_1-weighted Gd-enhanced image acquired 6 weeks after surgery. There is no change in the abnormal enhancement in the spinal cord. C: Sagittal T_2-weighted image obtained 46 months after surgery demonstrating the atrophic appearance of the spinal cord and small intramedullary hyperintensity at C5–6.

quences (Fig. 4A). Postoperative Gd-enhanced MR imaging clearly demonstrated white matter enhancement of the spinal cord at C5–6 (Fig. 4B). Follow-up MR imaging 3 months after surgery still demonstrated intramedullary hyperintensity, although the patient's clinical symptoms were improving.

Discussion

Histopathological studies indicate that intramedullary changes in chronic degenerative spinal disorders such as cervical spondylosis occur initially in the gray matter at the compression site. For example, Ito, et al.,[8] proposed that the first intramedullary pathological changes in spondylotic myelopathy are atrophy and neuronal loss in the anterior horn and intermediate zone. Such gray matter lesions are followed by degeneration of the lateral and posterior white columns. The authors of other clinicopathological studies[11,19,20] have also indicated that the white matter changes in cervical spondylosis were degenerative in nature and were considered to be irreversible. Intramedullary changes in cervical spondylosis or OPLL have been radiologically characterized as "fried egg–like"[9] enhancement on computerized tomography myelography or "snake-eye"[1] hyperintensity lesions on T_2-weighted MR imaging. It has been believed that these symmetrical abnormal findings in the spinal cord most likely represent microcystic degeneration of the gray matter.

In our series, abnormal intramedullary hyperintensity was revealed on T_2-weighted MR images both in the gray and white matter. The spinal cord at these levels had been slightly enlarged preoperatively and showed a more swollen appearance after decompressive surgery. Because the abnormal signal in the white matter resolved during the follow-up period, the hyperintensity was considered to be spinal cord edema caused by chronic compressive effects.

Magnetic resonance imaging enhancement of the spinal cord following administration of Gd has been reported in neoplastic lesions, demyelinating disorders,[14] acute transverse myelitis,[3,21] or acute spinal cord injury.[23,27] Although there have been no systemic studies involving Gd-enhanced MR imaging in patients with cervical spondylotic myelopathy, we found it unusual that the compressed spinal cord was enhanced by Gd in chronic degenerative spine disorders. Axial Gd-enhanced MR imaging revealed that the white matter was mainly enhanced in all patients. We speculate that disturbed venous circulation caused by

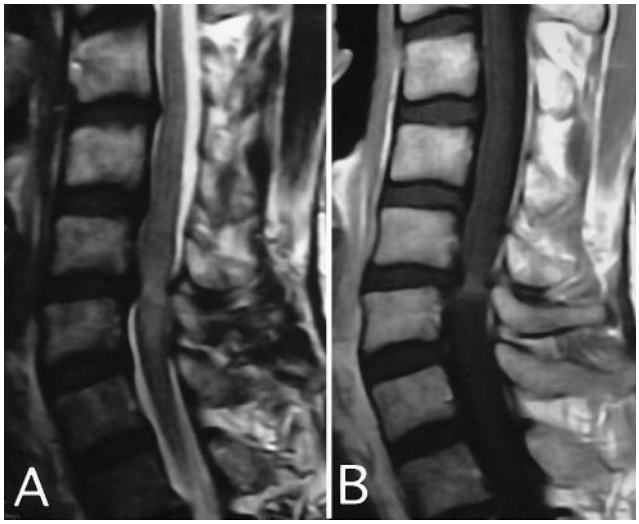

FIG. 3. Case 2. Preoperative MR images. A: Sagittal T_2-weighted image revealing spinal cord compression at C5–6 level; intramedullary hyperintensity is also visible at the same level. B: Sagittal T_1-weighted Gd-enhanced image demonstrating the intramedullary enhancement at C5–6.

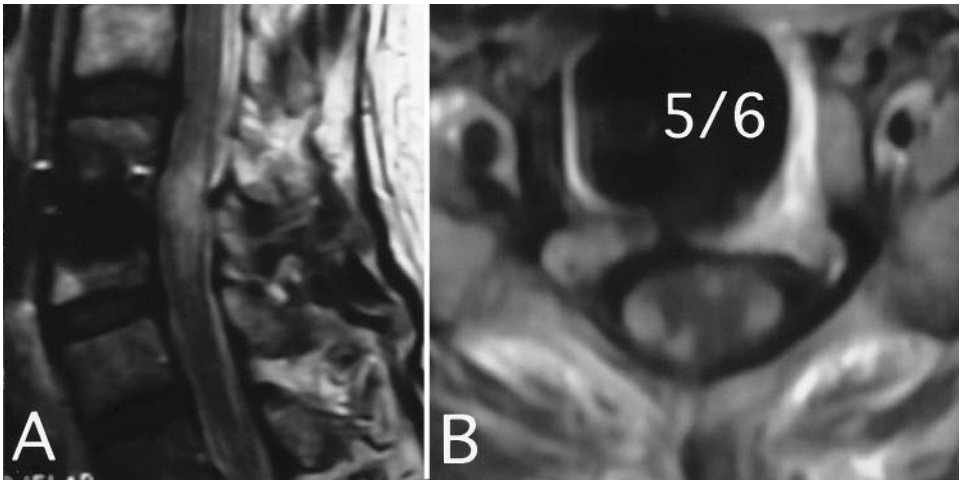

FIG. 4. Case 2. Postoperative MR images. A: Sagittal T_2-weighted image obtained 27 days after anterior fusion, demonstrating the increased intramedullary hyperintensity. B: Axial T_1-weighted Gd-enhanced images obtained at C5–6 (27 days postoperatively). The abnormal enhancement is mainly located in the white columns of the spinal cord.

spinal cord compression resulted in local venous hypertension at the affected level. Such venous hypertension would eventually result in venous ischemia or hyperpermeability of the intramedullary vessels and cause spinal cord edema at the compression site and adjacent levels. The presence of intramedullary Gd enhancement can be explained by the local hyperpermeability or a break of the brain–spinal cord barrier of the white matter vessels, most likely venous channels. Ischemia secondary to stenosis or occlusion at the arterial site was unlikely because of well-localized enhanced area and gradual progression of spinal cord symptoms. Occlusion of the anterior spinal artery would have caused more extensive lesions in the spinal cord and acute motor deficits.[25] Intramedullary Gd enhancement on MR imaging will involve mostly the gray matter in arterial spinal cord infarct.[6]

Disturbed CSF circulation may have played a role in the development of spinal cord edema in our series. Josephson, et al.,[10] reported spinal cord edema and intramedullary cyst formation in a rat model of spinal thecal sac constriction. Klekamp, et al.,[12] induced subarachnoid scarring by placing a kaolin-soaked fibrin sponge on the posterior surface of the feline spinal cord at C1–2. They found that the intramedullary pressure exceeded the subarachnoid pressure at C-2 in this condition. They concluded that the arachnoid scarring produced an interstitial type of edema in the central gray matter and enlarged perivascular space in the posterior columns because of altered CSF and extracellular fluid flow dynamics. Clinically, spinal cord edema has been reported in patients with Chiari malformation,[15] posterior fossa tumor, or postmeningitis state.[22] Fischbein, et al.,[5] reported five cases in which cervical myelopathy was secondary to various causes and in which enlargement of the spinal cord with T-1 and T-2 prolongation was revealed on MR imaging. These authors attributed obstruction of CSF pathways to the spinal cord enlargement with the intramedullary MR signal abnormality. Such a "presyrinx state" was improved by decompressive surgery in their series. Syringomyelia developing after surgical treatment of cervical spondylosis has also been reported,[18] although the exact pathomechanisms are unknown.

In our series, there were no visible subarachnoid spaces around the compressed spinal cord, as demonstrated on preoperative axial MR imaging in all six patients. Such subarachnoid space narrowing might cause the CSF blockage around the spinal cord and induce spinal cord edema or the presyrinx state. The spinal cord edema, however, was actually aggravated after decompressive surgery in our series. The intramedullary vascular changes, which were accompanied by a break of the blood–spinal cord barrier, may explain persistent postoperative spinal cord edema.

It is still unknown why the spinal cord edema—with the abnormal intramedullary Gd enhancement on MR imaging—deteriorated and persisted for several months after decompressive surgery in our series. In a retrospective study of 100 patients with cervical spondylotic myelopathy who underwent surgery, Ebersold, et al.,[4] described one patient with spinal cord edema and neurological deterioration 10 weeks after posterior decompression. The authors suggested spinal cord infarction as a cause of deterioration. Henderson, et al.,[7] reported a case of an extramedullary tumor at the C2–3 level in a patient in whom extensive spinal cord edema was documented after excision of the tumor. They attributed the cause of edema to obstruction of venous drainage of the spinal cord. Edema and white matter lesions have also been reported in venous congestion models of the spinal cord.[24,28] It is possible that a healing process of the hyperpermeability or a break of the blood–cord barrier of intramedullary vessels takes a long time to regain its previous functional state. Decompression of the spinal cord may induce acute reduction of the intravascular resistance and result in postoperative aggravation of spinal cord swelling.

Conclusions

We reported six cases of cervical spondylosis presenting with spinal cord edema at the compression level, which was aggravated after surgery. The spinal cord edema was caused by the reversible white matter lesion that most likely resulted from disturbed local venous cir-

culation. Such unusual MR imaging findings in cervical spondylotic myelopathy should be differentiated from intramedullary spinal cord tumors, demyelinating disorders, and inflammatory processes.

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