Clinical/Scientific Notes

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RARE INTRAMEDULLARY TANDEM LESION ON MRI DUE TO SPINAL ARTERIOVENOUS FISTULA

Spinal dural arteriovenous fistula (SDAVF) rarely presents as multiple signal-hyperintense intramedullary lesions on T2-weighted imaging, a finding more commonly seen in multiple neoplastic diseases like lymphoma or in demyelinating, inflammatory, infectious, or multilevel degenerative diseases. We report herein a case of SDAVF with tandem intramedullary hyperintense lesions on T2-weighted imaging.

Case report. A 58-year-old man with an 8-month history of worsening numbness in both legs progressing to gait disturbance within the next 2 months underwent MRI at another hospital. T2-weighted imaging revealed signal hyperintensities in spinal cord parenchyma at the Th6-8 level and conus medullaris. As no compressing spinal cord lesions were found, multiple sclerosis (MS) was suspected and he was referred to us to rule out intramedullary tumor. Neurologic findings were paraparesis (manual muscle test: right, 4/5; left, 3/5), left patellar hyperreflexia, bilateral hypesthesia and hypalgesia below Th6, position sense disturbance in both legs, dysesthesia at S1-2, and bowel and bladder dysfunction.

On T2-weighted imaging (figure, A), separate hyperintense lesions were found at Th6-8 and the conus medullaris. Lesions were isointense compared to normal spinal cord on T1-weighted imaging and displayed faint gadolinium enhancement (figure, B). T2-weighted imaging showed tiny, gadolinium-enhanced signal voids on the posterior spinal cord surface at Th8-10 (figure, A and B). Spinal angiography was performed on suspicion of SDAVF. As left Th7 intercostal arteriography (arterial phase) showed an enlarged perimedullary vein descending to the conus medullaris, SDAVF was diagnosed (figure, C). For spinal angiography below the Th2 level, all spinal levels were injected, revealing no early venous filling on additional arteriograms.

SDAVF was surgically interrupted after preoperative embolization. Paraparesis and sensory disturbance improved within 3 weeks. Signal hyperintensities on T2-weighted imaging were diminished at Th6-8 and the conus medullaris, far from the site of surgery. Signal void on the posterior spinal cord surface disappeared. After 9 months, no lesions were apparent on T2-weighted imaging (figure, D). No recurrence has occurred over the last 2 years, and the patient can walk with some left leg spasticity.

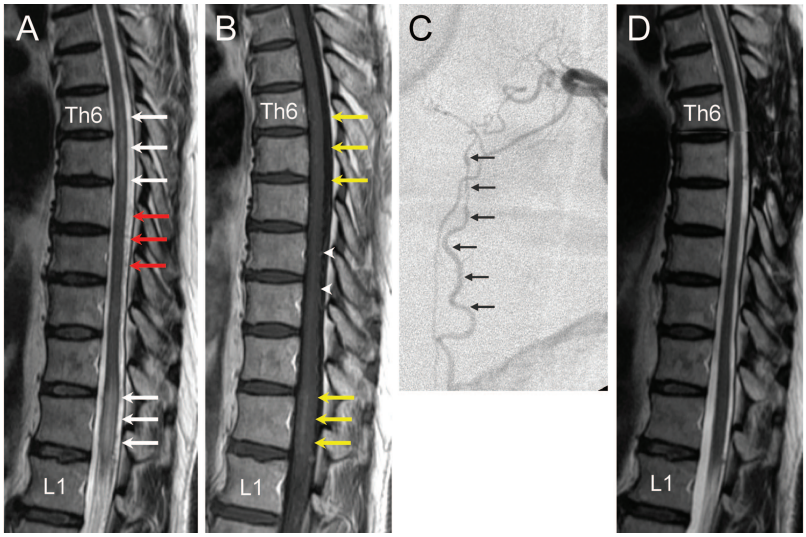
Discussion. Typically, T2-weighted imaging of SDAVF shows an abnormal intramedullary hyperintense lesion of the spinal cord and a serpiginous signal void on the spinal cord surface. On contrast-enhanced T1-weighted imaging, the signal void is enhanced and the spinal cord may also be enhanced. Venous-return disturbance results in spinal cord swelling and a high signal on T2-WI. The signal-hyperintense lesion is usually a single lesion, regardless of size. If multiple separate shunt points exist, multiple separate intramedullary lesions are seen. Previously reported multiple SDAVFs represented only a single continuous intramedullary high-signal lesion on T2-weighted imaging[1-3] and multiple intramedullary lesions due to a single SDAVF are rare.

Multiple intramedullary signal-hyperintense lesions on T2-weighted imaging result in a differential diagnosis of demyelinating disease such as MS, sarcoidosis, tuberculosis,[4] multiple neoplasms like lymphoma, hemangioblastoma in von Hippel–Lindau disease, and multilevel degenerative disease. As MRI has increased the detection rate of metastatic intramedullary tumors and multiple metastatic intramedullary tumors have been reported,[5] metastasis may be reflected in multiple intramedullary lesions.

The initial, incorrect diagnosis in our case was MS. Careful inspection disclosed a serpiginous signal void on the posterior spinal cord surface. The lesion was small and signal was low, reflecting low blood flow velocity. As signal void was missed on initial MRI, SDAVF was not considered as the cause of multiple intramedullary signal-hyperintense lesions on T2-weighted imaging.

We do not know why the single SDAVF in our case produced a T2-hyperintense lesion not only adjacent to the shunting point, but also in a distant caudal portion of the spinal cord. SDAVF symptoms and radiologic findings are due to increased venous pressure and impaired venous drainage from the spinal cord. The fistula produces venous blood flow

Figure Clinical course in images



(A) Intramedullary hyperintense lesions on T2-weighted MRI at the level of T6-8 and at the conus medullaris (white arrow). Note the tiny signal void at the posterior surface of the spinal cord between tandem lesions (red arrow). (B) T1-weighted MRI after IV injection of Gd-DTPA. T2-weighted signal-hyperintense lesions are faintly enhanced (yellow arrow). The signal void seen on the T2-weighted image is also enhanced (arrowhead). (C) Left Th7 intercostal arteriography, arterial phase. Early filling of an enlarged perimedullary vein descending on the surface of the spinal cord to the conus medullaris is recognized (black arrow). No obvious feeding artery can be seen. Diagnosis is spinal dural arteriovenous fistula. (D) T2-weighted MRI at 9 months postoperatively. Surgical interruption is only at the level of T6-7, but both lesions have completely disappeared.

stagnation and leads to impaired venous spinal cord drainage. Intradural arteriovenous malformation (AVM) has been postulated to have both rostral and caudal venous drainages, with this bidirectional blood flow preventing venous outflow stagnation and causing venous hypertension.[6] In contrast, peri- and intramedullary AVM near the conus medullaris may produce venous congestive myelopathy, since such lesions show only a superiorly directed venous outlet for major drainage routes such as DAVFs.[7] In our case, the conus lesion was distant from the shunting point. The conus may have provided unilateral drainage weaker than the bidirectional drainage afforded by the portion between lesions, and was thus selectively affected. Intraoperatively, we found a thrombosed vein. Although we cannot exclude the possibility that preoperative embolization produced this change, we posit that the tandem lesion was attributable to partial thrombosis.

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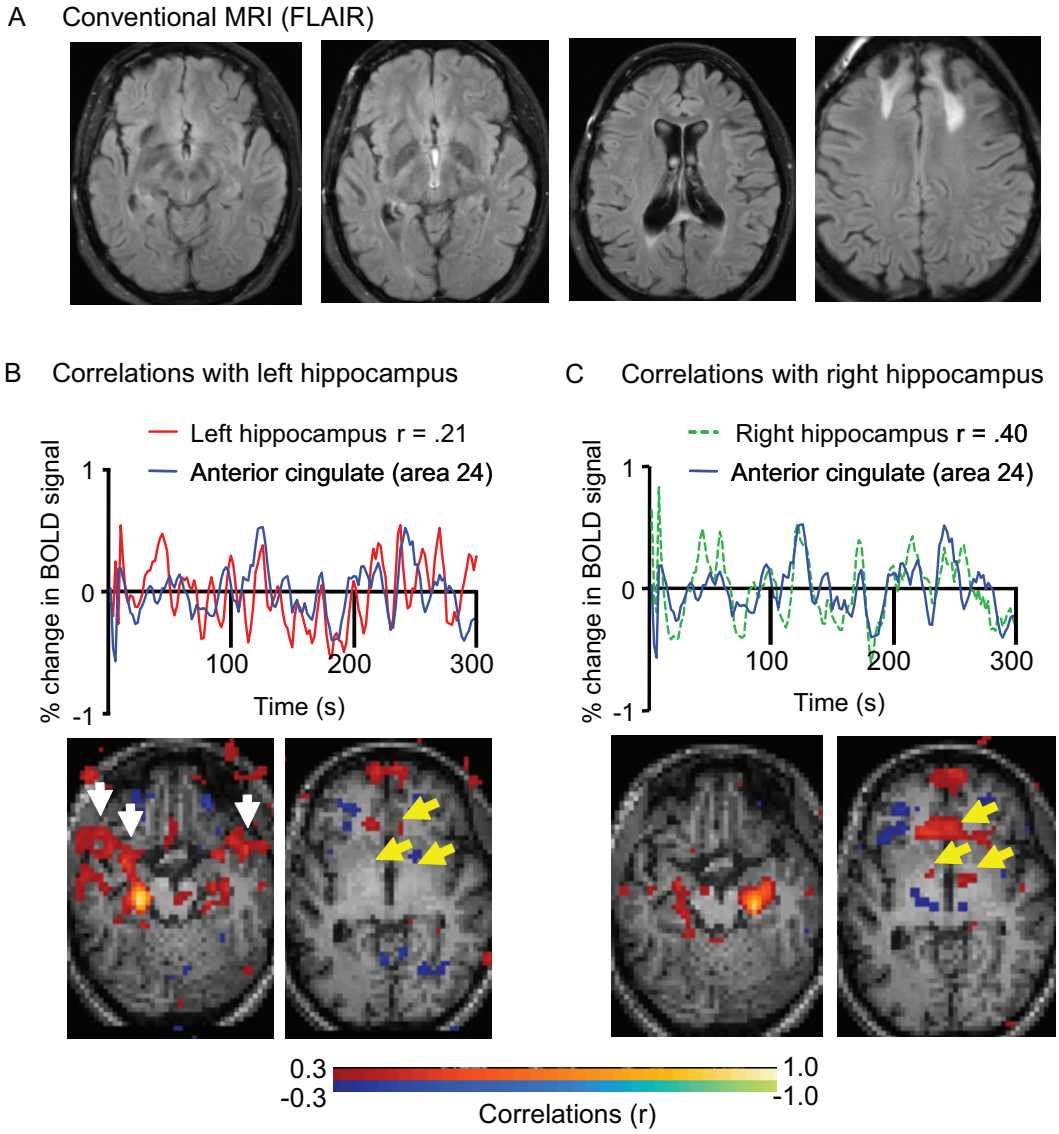
VERBAL MEMORY DEFICIT FOLLOWING TRAUMATIC BRAIN INJURY: ASSESSMENT USING ADVANCED MRI METHODS

Traumatic axonal injury resulting in disruption of functional networks in the brain is thought to be a major contributor to cognitive dysfunction in survivors of traumatic brain injury (TBI).[1] However, the true significance of this disruption is not known as current clinical imaging modalities do not directly assess axonal injury or functional network connectivity. Modern MRI techniques such as resting-state fMRI correlation analysis may overcome this limitation.[2,3]

A 21-year-old right-handed woman presented to the outpatient TBI clinic because she was "having trouble in school and remembering things." At 15, she was a passenger in a head-on motor vehicle collision. She had a severe TBI, clavicle fracture, and pulmonary contusions. Her initial Glasgow Coma Score was 4 (range: 3, deeply comatose, to 15, normal). Her initial head CT revealed a large left subdural hematoma with 1 cm left-to-right shift, bifrontal hemorrhagic contusions, subarachnoid hemorrhage, intraventricular hemorrhage, and punctate hemorrhages in the subcortical white matter, pons, and splenium of the corpus callosum. A left craniotomy was performed and the subdural hematoma was evacuated. She made a progressive neurologic recovery over 2 months. At discharge she was able to speak complete sentences and had no cranial nerve, motor, or sensory deficits. However, she has experienced

Figure Conventional MRI and resting-state fMRI correlation analysis in a 21-year-old with verbal memory deficits following traumatic brain injury



(A) Conventional MRI (FLAIR) revealed bilateral superior frontal lesions but no abnormalities that would explain the patient's verbal memory deficit (left to right: transverse slices at the level of hippocampus, thalamus, fornix, cingulum). (B) Resting-state fMRI correlation analysis revealed abnormal left hippocampal functional connectivity. Top panel: resting BOLD signal time course in the left hippocampus (red line) and anterior cingulate (blue line) were not well correlated ($r = 0.21$). Bottom panel: spatial map of resting BOLD correlations with the left hippocampus. Yellow arrows indicate absence of significant correlations between the left hippocampus and anterior cingulate or between left hippocampus and anterior thalamus. White arrows point to areas of abnormally increased correlation with the left hippocampus, of unknown importance. (C) Normal right hippocampal functional connectivity. Top panel: BOLD signal time course in the right hippocampus (green line) and anterior cingulate (blue line) were normally correlated ($r = 0.40$). Bottom panel: spatial map of resting BOLD correlations with right hippocampus. Significant correlations were observed between the right hippocampus and anterior cingulate as well as anterior thalamus (yellow arrows). Images displayed in anatomic space; patient's left side on the left side of the images.

persistent difficulties with memory and concentration affecting school performance and everyday life. Neuropsychological testing at age 21 revealed a moderately severe verbal memory deficit for auditory information but otherwise generally intact cognition (see supplementary data on the *Neurology*® Web site at www.neurology.org).

Conventional MRI scans of the brain revealed mild atrophy and bilateral superior frontal abnormalities with high T2 and FLAIR signal surrounding small regions of encephalomalacia (figure, A). These lesions were not thought to account for her memory loss. Brain regions associated with memory—specifically hippocampi, medial temporal lobes, thalami, and related white matter structures—were normal on conventional structural images.

Quantitative hippocampal volumetry[4] revealed that the left hippocampus was 18% smaller than the right (3,538 mm^3 vs 4,185 mm^3). Although each side individually was within the range of 10 normal controls (3,516 to 4,303 mm^3), this degree of hippocampal asymmetry was abnormal; absolute right to left difference in controls was 1–10%.

Diffusion tensor imaging revealed subtle abnormalities in the left and right cingulum bundles as well as left and right fornices. This technique measures the diffusion of water in many directions. It is sensitive to axonal injury as it detects microstructural abnormalities which reduce directional anisotropy of water diffusion.[5] Relative anisotropy in the cingulum and fornix were lower than the mean of 10 control subjects matched in age, gender, and handedness. However, relative anisotropy was not more than 2 standard deviations below the mean in any of the regions, and so cannot be considered definitively abnormal (see supplementary data).

Thus, it was initially not clear whether the lower left hippocampal volume and borderline low relative anisotropy in the cingulum and fornix were functionally significant. Resting state fMRI correlation analysis, however, clearly demonstrated disruption of the normal connectivity of the left hippocampal network (figure, B). Specifically, this analysis revealed that the BOLD fluctuations in the left hippocampus correlated poorly with those in several other structures implicated in memory including anterior thalamus and the ventral anterior cingulate cortex (vACC). In contrast, the fluctuations in the right hippocampus were normally correlated with those in these structures (figure, C). No such left-right asymmetry in the hippocampal-anterior thalamic and hippocampal-vACC correlations was seen in 10 age-matched controls (not shown).

From this, we conclude that the patient had a functional disruption of the left hippocampal network. This is likely responsible for her verbal memory deficit.[6] We hypothesize that the combined effects of her three relatively mild lesions (to the left hippocampus, fornix, and cingulum bundle) caused the disruption. The cingulum bundle injury may have been due to subfalcine herniation, as this region is not commonly affected by traumatic axonal injury. Traumatic axonal injury in the fornix, instead, has been frequently reported,[4,7] as has post-traumatic hippocampal volume loss.[4]

Resting-state fMRI correlation analysis appears to be a remarkably powerful tool for assessing the sequelae of TBI. This technique may be especially helpful in clarifying the functional significance of subtle anatomic abnormalities uncovered using other imaging methods such as DTI and quantitative volumetry. The general utility of this combined advanced MRI approach for diagnosis, prognosis, rehabilitative planning, and therapeutic trial stratification will be the subject of future investigations.

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