Diffusion Tensor Fiber Tractography for Arteriovenous Malformations: Quantitative Analyses to Evaluate the Corticospinal Tract and Optic Radiation

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BACKGROUND AND PURPOSE: We hypothesized that diffusion tensor fiber tractography would be affected by intracranial arteriovenous malformation (AVM). The purpose of the present study was to evaluate the influence of intracranial AVM on corticospinal tract and optic radiation tractography.

MATERIALS AND METHODS: The subject group comprised 34 patients with untreated intracranial AVM. Hemorrhage was present in 13 patients and absent in 21 patients. Perinidal fractional anisotropy (FA) and number of voxels along the reconstructed corticospinal and optic radiation tracts were measured, and left-to-right asymmetry indices (Als) for those values were quantified. Patients were assigned to 1 of 3 groups: tracts distant from nidus, tracts close to nidus without neurologic symptoms, and tracts close to nidus associated with neurologic symptoms. One-way analysis of variance was used to compare differences in Al between groups. Hemorrhagic and nonhemorrhagic groups were assessed separately.

RESULTS: In patients without hemorrhage, Al of optic radiation volume (P < .001), Al of perinidal FA along corticospinal tract (P = .006), and optic radiation (P = .01) differed significantly between groups. In patients associated with hemorrhage, Al of corticospinal tract volume (P = .01), Al of perinidal FA along corticospinal tract (P = .04), and optic radiation (P = .004) differed significantly between groups.

CONCLUSIONS: Corticospinal tract and optic radiation tractography were visualized in patients with AVM. In patients with both hemorrhagic and nonhemorrhagic AVM, the 2 fiber tracts close to the nidus were less visualized in the affected hemisphere than those distant from the nidus. Tracts were less visualized in patients with neurologic symptoms than in asymptomatic patients.

Subjects and Methods

Patients

MR imaging was performed on 41 consecutive patients with intracranial AVM who presented to our institution between May 2004 and December 2005. Of these 41 patients, 6 patients were excluded because of metal artifact near the AVM nidus as a result of previous treatment, and another patient was excluded because of poor image quality attributable to acute large intracerebral hemorrhage and bulk motion. The subject group comprised the remaining 34 consecutive patients (18 men and 16 women; mean age, 34 years; range, 6–72 years) with intracranial AVM. All of the patients were diagnosed with AVM on the basis of cerebral angiography. Institutional review board approval was obtained for this study. Written informed consent was obtained from all of the adult patients and from the parents of the 4 pediatric patients. According to the Spetzler-Martin grading system, patients without hemorrhage were classified as follows: grade 1, 4 patients; grade 2, 9 patients; grade 3, 5 patients; grade 4, 3 patients; and grade 5, no patient. Similarly, patients associated with hemorrhage were as follows: grade 1, 1 patient; grade 2, 6 patients; grade 3, 4 patients; grade 4, 1 patient; and grade 5, 1 patient. Two experienced neurosurgeons performed neurologic examinations in all of the patients, including manual muscle testing and visual field examination, by using confrontation method. Patients suspected of visual field defect were confirmed with the use of Goldmann perimetry. Details of patient demographics are provided in Table 1.

Data Acquisition

MR imaging was performed using a whole-body 3T MR scanner (Trio; Siemens, Erlangen, Germany) equipped with a 40 mT/m gradient, integrated parallel acquisition technique, and a receiver-only
Table 1: Demographic data for 34 patients with untreated intracranial AVM

| Patient No. | Sex | Age at MR Imaging (years) | Location of Nidus | Hemorrhage | Perifocal Hyperintensity | Neurologic Symptoms | Spetzler-Martin Grade |
|-------------|-----|---------------------------|-------------------|------------|-------------------------|--------------------|---------------------|
| 1           | F   | 29                        | Right occipital   | No         | No                      | No                 | 2                   |
| 2           | M   | 31                        | Right frontal     | No         | No                      | No                 | 1                   |
| 3           | F   | 27                        | Left occipital    | No         | Yes                     | Right homonymous hemianopsia | 3                   |
| 4           | M   | 64                        | Right occipital   | No         | No                      | No                 | 2                   |
| 5           | M   | 49                        | Left frontal      | No         | Yes                     | No                 | 2                   |
| 6           | M   | 72                        | Left parietal     | No         | No                      | No                 | 1                   |
| 7           | M   | 25                        | Left occipital    | No         | No                      | No                 | 2                   |
| 8           | M   | 45                        | Right frontal     | No         | Yes                     | No                 | 3                   |
| 9           | M   | 28                        | Left frontal      | No         | No                      | No                 | 3                   |
| 10          | M   | 49                        | Left putamen      | No         | No                      | No                 | 2                   |
| 11          | M   | 25                        | Right temporal    | No         | Yes                     | No                 | 2                   |
| 12          | M   | 28                        | Right occipital   | No         | No                      | Left lower quadranopsia | 4                   |
| 13          | F   | 5                         | Left parietal     | No         | No                      | No                 | 1                   |
| 14          | M   | 59                        | Right frontal     | No         | Yes                     | No                 | 1                   |
| 15          | F   | 28                        | Right parietal    | No         | No                      | No                 | 2                   |
| 16          | M   | 45                        | Left cingulum     | No         | No                      | No                 | 2                   |
| 17          | F   | 33                        | Right cingulum    | No         | No                      | No                 | 4                   |
| 18          | F   | 29                        | Left frontal      | No         | No                      | No                 | 2                   |
| 19          | M   | 28                        | Right insula      | No         | No                      | No                 | 4                   |
| 20          | F   | 17                        | Left cerebellum   | No         | Yes                     | Cerebellar ataxia  | 3                   |
| 21          | F   | 20                        | Left frontal      | No         | No                      | No                 | 3                   |
| 22          | F   | 45                        | Right occipital   | Yes        | No                      | No                 | 3                   |
| 23          | F   | 29                        | Left occipital    | Yes        | No                      | No                 | 2                   |
| 24          | M   | 22                        | Right cerebellum  | Yes        | No                      | Cerebellar ataxia  | 4                   |
| 25          | F   | 15                        | Right occipital   | Yes        | No                      | No                 | 3                   |
| 26          | F   | 56                        | Right cerebellum  | Yes        | Yes                     | Cerebellar ataxia, right trigeminal palsy | 1                   |
| 27          | M   | 45                        | Right parietal    | Yes        | Yes                     | Recent memory disturbance | 3                   |
| 28          | F   | 21                        | Right occipital   | Yes        | Yes                     | Left upper quadranopsia | 2                   |
| 29          | F   | 27                        | Right parietal    | Yes        | Yes                     | Left hemianopsia   | 2                   |
| 30          | F   | 6                         | Right frontal     | Yes        | Yes                     | Left hemiparesis (MMT4/5) | 2                   |
| 31          | M   | 38                        | Right parietal    | Yes        | No                      | Left hemiparesis (MMT4/5) | 2                   |
| 32          | M   | 50                        | Left frontal      | Yes        | Yes                     | Motor aphasia      | 5                   |
| 33          | F   | 23                        | Right frontal     | Yes        | Yes                     | Left hemiparesis (MMT4/5) | 2                   |
| 34          | M   | 48                        | Left parietal     | Yes        | Yes                     | Right hemiparesis (MMT3/5), right spatial neglect | 3                   |

Note: M indicates male; F, female; MMT, manual muscle testing. Patients 1–21 were without intracranial hemorrhage, and patients 22–34 displayed hemorrhage.

8-channel phased-array head coil. A single-shot spin-echo echo-planar sequence was applied for DT imaging with the following parameters: TR, 7000 ms; TE, 79 ms; motion-probing gradient in 40 non-colinear directions and 4 b values: TR, 700 s/mm²; matrix, 128 × 104; voxel size, 2 × 2 × 2 mm; no intersection gap; and single averaging. The generalized autocalibrating partial parallel acquisition (GRAPPA) algorithm was applied for parallel imaging, with a reduction factor of 2 and 24 additional autocalibrating phase-encoding lines in the center of k-space. Section planes and field center were the same as in DT imaging. Acquisition time was 5 minutes and 30 seconds.

Whole-brain MR angiography was also obtained to identify the AVM nidus. The 3D time-of-flight technique was applied with the following parameters: TR, 22 ms; TE, 3.9 ms; flip angle, 20°; 5-slab acquisition with 44 sections per slab and 25% section oversampling; section thickness, 0.64 mm; matrix, 320 × 256; and voxel size, 0.63 × 0.63 × 0.64 mm. The GRAPPA algorithm was also applied for parallel imaging, with a reduction factor of 2 and an additional 36 autocalibrating phase-encoding lines in the center of k-space. Section planes and field center were the same as in DT imaging. Acquisition time was 4 minutes 8 seconds.

**DT Imaging Data Processing and Fiber Tractography Reconstruction**

DT imaging datasets were transferred in Digital Imaging and Communications in Medicine format to a Windows personal computer workstation. DTIStudio version 2.02 software (H. Jiang and S. Mori, Department of Radiology, Johns Hopkins University, Baltimore, Md) was used for tensor calculations. The details of DT imaging data processing were described elsewhere. Three eigenvalues and eigenvectors were obtained, and then fractional anisotropy (FA) maps and a directional color-coded map were synthesized.

Fiber tractography was performed on the basis of the fiber assignments derived by continuous tracking (FACT) method. Tracking from all of the pixels inside the brain (ie, by using the brute force approach) was performed, initiated in both retrograde and orthograde directions according to the direction of the principal eigenvector in each voxel. Results that penetrated the hemisphere could be reconstructed. When multiple ROIs were used for tract reconstruction, 3 types of operation were applied: AND, OR, and NOT. Choice of operations depended on the characteristic trajectory of the tract. Propagation in each fiber tract was terminated if a voxel with an FA value of <0.2 was reached or if the turning angles of 2 consecutive vectors were >70° during tracking. A relatively large angle threshold was used so that the tract would not turn sharply.

To reconstruct corticospinal tractography, 2 ROIs were seg-
One author performed all of the ROI segmentations. The first “OR” ROI (white polygon) is placed at either side of the occipital lobe, including the calcarine cortex on the coronal plane through the anterior edge of the occipital-parietal sulcus (top left). The second “AND” ROI (blue) is placed on the ipsilateral precentral gyrus (top right). Left and right ROI segmentations were separately performed. “NOT” ROIs (green) are placed on midline structures connecting right and left corticospinal tracts on sagittal reconstructed image (bottom left) and fibers projecting into ipsilateral cerebellar peduncle on coronal reconstructed image (bottom right). Examples of bilateral corticospinal tract overlaid on coronal reconstructed image are shown (bottom right).

**Fig 1.** A, ROI segmentation for corticospinal tract tractography. Polygonal ROIs are placed on transverse $b = 0$ images (TR/TE, 7000 ms/79 ms). The first “OR” ROI (white polygon) is placed at either side of the cerebral peduncle on the plane where the characteristic $\Omega$ shape of the central sulcus is at the center of cerebral hemisphere (top left). The second “AND” ROI (blue) is placed at the ipsilateral precentral gyrus (top right). Left and right ROI segmentations were separately performed. “NOT” ROIs (green) are placed on midline structures connecting right and left corticospinal tracts on sagittal reconstructed image (bottom left) and fibers projecting into ipsilateral cerebellar peduncle on coronal reconstructed image (bottom right). Examples of bilateral corticospinal tract overlaid on coronal reconstructed image are shown (bottom right). B, ROI segmentation for optic radiation tractography. The 4 kinds of ROIs (white polygon) are placed on coronal or sagittal color-coded maps. Cross lines indicate the orthogonal planes. The first “OR” ROI is placed at either side of the occipital lobe, including the calcarine cortex on the coronal plane through the anterior edge of the occipital-parietal sulcus (top left). The second “AND” ROI is placed at the ipsilateral temporal stem, including the Meyer loop on the sagittal plane (top right). Temporal stem is identified as green, and the Meyer loop is identified as a small red area inside the temporal stem (red arrow). The third and fourth “NOT” ROIs are placed on the same coronal plane through the dorsal end of the Sylvian fissure (bottom left). Bilateral Meyer loops are indicated as red arrows. Occipital-frontal connections medial to the Meyer loop and fibers projecting to the temporal horn passing through lateral to the Meyer loop are removed using the “NOT” operation. Examples of bilateral optic radiation overlaid on transverse $b = 0$ images (TR/TE, 7000 ms/79 ms) are shown (bottom right).

**Data Analysis for Fiber Tractography**

The distances between the margins of tractography and the margin of the AVM lesion (including nidus, hematoma, and draining vein) were measured on transverse $b = 0$ images. Sections for measurement were manually selected by 1 of the authors, where tractography appeared to be nearest to the margin. When the nidus was too small to be identified on $b = 0$ images $(n = 3$; Spetzler grade 1 AVM, $n = 1$ [patient 13]; Spetzler grade 2 AVM, $n = 2$ [patients 5 and 10]), images from MR angiography were referred. Subjects were assigned to 1 of the following groups according to measured distances and the presence of neurologic symptoms: group A, tracts distant from nidus; group B, tracts close to nidus without neurologic symptoms; and group C, tracts close to nidus associated with neurologic symptoms. Distances $\geq 1$ cm between tracts and nidus were assigned “tracts distant from nidus” and $< 1$ cm were assigned “tracts close to nidus.” Motor weakness in the contralateral side was defined as neurologic symptom related to corticospinal tract damage, and visual field defect presented as contralateral hemianopsia or quadrantanopsia was defined as related to optic radiation damage. A group of representative patients are shown in Fig 2A (corticospinal tract) and Fig 2B (optic radiation). In these figures, depiction of tractography, vasculature, and lesion in 3D were performed by using Amira 4.0 software (Mercury Computer Systems, Chelmsford, Mass).

Various changes in tractographic appearances were observed in affected hemispheres. Tractographic appearances were classified into 4 categories: no change, compression, penetration, and disruption. Visually intact tract displaced by the lesion was defined as compression, intact tract propagating through the dilated nidal vessels was defined as penetration, and tract terminated near the lesion was de-
The left optic radiation was disrupted around the occipital pole. The number of voxels along the tractography reportedly displays little asymmetry. To quantitatively evaluate what asymmetrical, corticospinal tract tractography in healthy subjects displays, 2 parameters were selected: FA of perinidal region and number of voxels along the tractography. Corticospinal tract damage was reported to correlate with FA along the tractography in patients with amyotrophic lateral sclerosis or chronic ischemic stroke. The number of voxels along the tractography represented the entire volume of tractography. These parameters were recorded by using a specific function of DTI Studio. Two ROIs were placed on the FA map, one in the tract located nearest to the AVM nidus, and the other in the contralateral tract of the same section. ROI for the corticospinal tract was segmented on an axial FA map. Each ROI has 9 oval-shaped 36-mm² pixels.

Although diffusion characteristics in the normal brain are somewhat asymmetrical, corticospinal tract tractography in healthy subjects reportedly displays little asymmetry. To quantitatively evaluate mean FA and number of voxels along the tractography in affected hemispheres, asymmetry index (AI) of the 2 parameters between the affected and unaffected hemispheres was calculated by applying the following equation: the difference in parameters between unaffected and affected sides was divided by the mean of both sides \( AI = \frac{(unaffected - affected)}{(unaffected + affected)/2} \), as described previously. Index range was from −2 to 2.

For statistical analysis, 1-way analysis of variance was performed to compare differences in AIs of the mean FA and number of voxels along the tractography between each distance score. Hemorrhagic and nonhemorrhagic patients were assessed separately. Statistical analyses were performed by using JMP5.1 software (SAS Institute, Cary, NC). Values of \( P < 0.05 \) were considered statistically significant.

**Results**

Corticospinal tract tractography was visualized in all of the patients. Regarding the distance between lesion and tractography in the affected hemisphere and motor weakness, results for patients without hemorrhage were as follows: group A, \( n = 14 \); group B, \( n = 7 \); and group C, \( n = 0 \). No patient with AVM close to corticospinal tract presented motor weakness. Among the 7 patients assigned to group B, corticospinal tract was compressed medially in 1 patient (patient 17), and the other patients displayed no remarkable shift or disruption. Similarly, results for patients associated with hemorrhage were as follows: group A, \( n = 7 \); group B, \( n = 2 \); and group C, \( n = 4 \). Corticospinal tract of 1 patient assigned to group B was compressed anteriorly (patient 29), and the other patient displayed no remarkable shift or disruption. Among the 4 patients assigned to group C, corticospinal tract in the affected hemisphere was veering laterally at the level of the centrum semiovale, and projection fibers from the medial precentral gyrus were not visualized. The corticospinal tract was compressed posteriorly in patient 33 and no remarkable shift or disruption. Among the 4 patients assigned to group C, corticospinal tract was compressed posteriorly in patient 33 and was not shifted or disrupted in the remaining 2 patients.

Optic radiation tractography was also visualized in all of the patients. Results for patients without hemorrhage were as follows: group A, \( n = 14 \); group B, \( n = 5 \); and group C, \( n = 2 \). Among the 5 patients assigned to group B, 2 tracts were com-
pressed by the nidus (patients 1 and 15), 2 were disrupted by the nidus (patients 4 and 7), and the remaining patient displayed no remarkable shift or disruption. Optic radiation of one patient assigned to group C was disrupted (patient 3), and the other patient displayed no remarkable shift or disruption. Similarly, results for patients associated with hemorrhage were as follows; group A, n = 7; group B, n = 3; and group C, n = 3. Among the 3 patients assigned to group B, optic radiation was compressed by the hemorrhagic nidus in patient 22, was penetrating the hemorrhagic nidus in patient 25, and was not shifted or disrupted in another patient. Among the 3 patients assigned to group C, optic radiation was compressed by the hemorrhagic nidus in patients 29 and 34 and was not shifted or disrupted in another patient. Tractographic appearances are summarized in Table 2.

In patients without hemorrhage, AI of optic radiation volume (P < .0001), AI of perinidal FA along corticospinal tract (P = .006), and optic radiation (P = .01) differed significantly between groups. AI of corticospinal tract volume did not differ significantly between groups. In patients with hemorrhage, AI of perinidal FA (P = .04), tract volume (P = .01) of corticospinal tract, and AI of perinidal FA along optic radiation (P = .004) differed significantly between groups. AI of optic radiation volume did not differ significantly between groups. Statistical results are summarized in Fig 3.

**Discussion**

The present study visualized the corticospinal tract and optic radiation tractography in patients with AVM and assessed the influence of intracranial AVM on tractography. DT imaging and fiber tractography techniques enable in vivo visualization of eloquent fiber tracts, a feat that is virtually impossible to achieve using conventional MR imaging techniques. Both 2D presentation of voxels where the fiber tract penetrates the section and 3D presentation of all of the tractographic images are useful for visualizing course changes of the fiber tract (Fig 2A, B). Clinical application of tractography depends on the visualization of tractography in patients with intracranial space occupying the lesion. Our results show that, in patients without hemorrhage, perinidal FAs of both corticospinal tract and optic radiation were significantly different between the groups, and tract volume was significantly different only for the optic radiation between the groups. No patient experienced motor weakness, and no patient presented corticospinal tract disruption. One patient experienced right homonymous hemianopsia with disrupted optic radiation, though another
patient experienced lower left quadrantanopsia without disruption. The 2 other patients with disrupted optic radiation did not complain of any visual field defects.

In patients with brain tumor, corticospinal tract tractography has reportedly been disrupted as a result of tumor compression, direct involvement, or peritumoral edema. In our results, perinidal FA along the 2 fiber tracts and optic radiation volume were significantly different between the 3 groups, though corticospinal tract volume did not significantly differ between the groups. In patients without neurologic deficit, corticospinal tract abutting the nidus was not disrupted in any patient, but optic radiation disruption abutting the nidus did not necessarily reflect visual field defect. Although nonhemorrhagic AVM has much less vasogenic edema, which seems to decrease diffusion anisotropy and subsequent FA values, our results show decreased perinidal FA in patients with nonhemorrhagic AVM. FA values of the eloquent fiber tracts near the nidus may reflect a change of microvasculature in normal appearing white matter near the nidus. The different results from brain tumor and AVM are probably due to the pathologic nature of these 2 lesions. Even if the nidus could be deeply localized, particularly in the watershed lesions, AVM feeders are frequently superficial, and for the most part the actual vascular lesions are not infiltrating the white matter tract. The reasons for different results from the corticospinal tract and optic radiation tractography were probably related to technical difficulties in optic radiation tractography, as explained later in detail. Differences in anatomic features in the 2 fiber trajectories might also play a role. The corticospinal tract follows an anatomically coherent, colinear course between the centrum semiovale and cerebral peduncle. Conversely, the optic radiation is defined as a geniculo-cortical tract that begins at the lateral geniculate body, follows the lateral wall of the lateral ventricle into the temporal lobe, and terminates at the striate area on the medial surface of the occipital lobe on either side of the calcarine sulcus. However, course in the temporal lobe is intermingled with other fibers that course in various directions and are indistinguishable from surrounding fibers. The optic radiations are not as coherent as corticospinal tract tractography, and these anatomic differences might have affected the results.

Hemorrhage associated with AVM induces vasogenic edema and susceptibility effects, and technical difficulties are considered likely to occur when performing tractography because of signal intensity drop-off in DT imaging, which is more prominent at 3T than at 1.5T. In the present cases with hemorrhagic AVM (n = 13), perinidal FA along the 2 fiber tracts and corticospinal tract volume were significantly different among the 3 groups, though optic radiation volume did not significantly differ among the groups. Four patients experienced motor weakness, including one patient whose corticospinal tract was veering laterally; fibers projecting from the medial precentral gyrus were disrupted; and the corticospinal tracts of the remaining 3 patients were not disrupted. No patient displayed disrupted optic radiation, though 3 patients suffered visual field defect or spatial neglect. These results are partly compatible with previous reports of white matter FA change in patients with brain tumor or cerebral hemorrhage. In our subjects, 8 patients had perifocal hyperintensity among the 13 hemorrhagic patients, which showed higher prevalence than nonhemorrhagic patients. Perifocal hyperintensity, including edema or gliosis, may have caused the FA change in the hemorrhagic subjects. Visually demonstrated “tract disruption” abutting the nidus did not necessarily reflect neurologic symptoms.

The present study reveals that perinidal FA and tract volume along the tract close to an AVM nidus are significantly lower than in the contralateral hemisphere. In contrast, tractographic appearance of “disruption” does not correlate with clinical symptoms, especially in patients associated with hemorrhage. Both the asymptomatic patient with disrupted tract (false-positive) and the symptomatic patient without tract disruption (false-negative) were present. This is probably due to the technical limitation of DT imaging and tractography induced by artifacts, including susceptibility effects. A decrease in perinidal FA along the tract or in tract volume reflects white matter change and subsequent clinical symptom more reliably than changes in tractographic appearances. Although clinical applications of tractography, such as functional evaluation or presurgical planning, should be performed carefully, relationships between eloquent fibers and AVMs have been visualized in patients with both hemorrhagic and nonhemorrhagic lesions. Attempts to validate the location of corticospinal tract tractography and optic radiation tractography compared with electrophysiologic testing for motor or visual function are still under way.

Three major therapeutic strategies have been applied for intracranial AVM treatment: surgical operation, stereotactic radiosurgery, and endovascular embolization. Surgical treatment for intracranial AVM is challenging, given the need to manage obliteration of the nidus in addition to risk of intraoperative hemorrhage and postoperative edema or hemorrhage because of normal perfusion pressure breakthrough. AVM operations are special in that palliative resection increases the risk of bleeding inversely in relation to the operation, and complete resection has always been required for surgical management of intracranial AVM, whereas other intracranial space-occupying lesions receive partial resection to avoid postoperative neurologic deficits. Surgical indications for AVMs should thus be determined by considering the risk of postoperative deficits after complete resection. Locations of motor and visual pathways are more important for determining surgical indications of AVM near eloquent brain areas than other intracranial space occupying lesions, because an “all-or-nothing” operation is required for this condition. According to our results, both corticospinal tract and optic radiation tractography are supposed to be clinically feasible in considering the spatial distribution of eloquent fibers, though the correlation between tractographic appearance and clinical condition should be carefully interpreted. If surgical obliteration of an AVM is considered high risk because of the relationship between the lesion and eloquent white matter and eloquent cortices, alternative therapeutic strategies might be applied, such as stereotactic radiosurgery, endovascular embolization, or a combination of embolization and surgery. DT tractography has been reported to be useful in confirming the radiation dose to the corticospinal tract during radiosurgical planning and thus provides information for both therapeutic strategies and detailed planning.

The present study includes some limitations. First, techni-
cal difficulties in tractography may have affected the results. We applied a multiple ROI approach by using “OR,” “AND,” and “NOT” operations of choice. Although this approach is useful for known 3D configuration of white matter tract, and good agreement with postmortem anatomic studies has been reported,2,27 “NOT” operations may lead to spurious elimination of tracts. At present, no tractography technique based on DT datasets visualizes fiber tracts of interest without any subjective ROI operations. Methods with greater precision and reproducible outcomes will be required in the future. Some differences in corticospinal tract tractography and optic radiation tractography also should be noted. Unlike cases with corticospinal tract, optic radiation tractography is more challenging. A number of authors have attempted to visualize the optic radiation tractography in different ways,7,13,28,29 and no “gold standard” method has been established. No quantitative analysis methods for optic radiation tractography have yet been described. Measured parameters and analysis methods were based on the studies for corticospinal tract tractography, and the feasibility of parameters may remain questionable in optic radiation tractography. The second limitation is that tractography reconstruction is not a precise stepwise procedure with perfectly reproducible outcomes but is somewhat dependent on ROI manipulation and tracking algorithms. The FACT algorithm applied in this study is susceptible to a phenomenon known as “crossing fiber problems.” New fiber-tracking algorithms have been applied to resolve such problems, and future technical advances will bring these new algorithms into clinical use. Optic radiation tractography by using probabilistic tractography techniques has been reported,28 and this method might improve the difficulties associated with optic radiation tractography in the present study.

Conclusions
In patients with either hemorrhagic or nonhemorrhagic AVM, corticospinal tract and optic radiation tractography close to the nidus were less visualized in the affected hemisphere than those distant from the nidus. Tracts were less visualized in patients with neurologic symptoms than in asymptomatic patients. Fiber tractography for intracranial AVM visualizes relationships between eloquent fibers and AVM, and the information provided from eloquent white matter by fiber tractography may be useful for therapeutic strategies.

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