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CASE REPORT

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SUMMARY: It is unknown whether dilated perivascular spaces can affect the adjacent neuronal fibers. We describe conventional MR and diffusion tensor imaging findings of a case with multiple, prominent dilated perivascular spaces in the left cerebral hemisphere. Diffusion tensor imaging showed no alterations in the fractional anisotropy and apparent diffusion coefficient values for the corona radiata, posterior rim of the internal capsule, and the cerebral peduncle, indicating no wallerian degeneration associated with dilated perivascular spaces.

Brain perivascular spaces (PVSs) are pial-lined, interstitial fluid-filled structures that accompany penetrating arteries and arterioles for a variable distance as they descend into the cerebral substance.¹ It is unknown whether prominent dilated PVSs can affect the neuronal fibers of the white matter. Many authors described the presence of hyperintense areas in the adjacent white matter of dilated PVSs on fluid-attenuated inversion-recovery (FLAIR) images, but their cause is unknown.²⁻⁴ If dilated PVSs affect the neuronal fibers of the white matter, neuronal degeneration such as wallerian degeneration may be seen; diffusion tensor imaging (DTI) is more sensitive for the detection of the neuronal degeneration than conventional MR imaging methods.⁵⁻⁹ Here, we present DTI findings in a case with multiple, prominent dilated PVSs involving a single cerebral hemisphere.

Case Report

A 55-year-old man underwent “brain dock” (a screening of the brain practiced in Japan upon a person’s demand for a medical examination) with MR imaging. He had no neurologic abnormality, no previous illness, and no significant family history. Because the MR images showed abnormal findings, and brain ischemia could not be ruled out as the cause, the referring physician sent him to our hospital for further evaluation. Here he underwent MR imaging, including DTI and MR angiography. MR angiography was performed with a 3D time-of-flight technique for head and cervical regions. All MR imaging scans were obtained with a 1.5T superconducting system (Magnetom Vision; Siemens, Erlangen, Germany). The DTI MR imaging was obtained using a Stejskal-Tanner sequence with single-shot, spin-echo-type echo-planar imaging, a flip angle of 90°, and a repetition time of 4000 ms. The echo time was 100 ms. The matrix size was 128 × 128, and the field-of-view was 220 × 220 mm. Twenty sections, 5 mm thick with no gap, were obtained. Motion-probing gradients in 6 orientations were applied after acquisition of b = 0 images. The parallel imaging technique was not applied. The brain fiber tracking, color-coded map, and calculation of apparent diffusion coefficient (ADC)

and fractional anisotropy (FA) were performed using free software (dTV version 1.2) for DTI analysis developed by the Image Computing and Analysis Laboratory, Department of Radiology, The University of Tokyo, Japan.

Axial T1- and T2-weighted spin-echo and fast FLAIR images showed multiple, well-defined, rounded areas of various sizes, predominantly in the periphery of the left cerebral hemisphere, with signal intensity identical to that of CSF (Fig 1A). The FLAIR images revealed hyperintense foci adjacent to these dilated PVSs in the left cerebral white matter, but no notable foci on the right cerebral hemisphere were evident (Fig 1A). There was no apparent atrophy in any brain structures. On MR angiography, no abnormality was seen in the cervical carotid and intracranial arteries. ADC maps demonstrated increased diffusion similar to CSF for the dilated PVSs. FA maps showed decreased FA in the periphery of the left cerebral white matter (Fig 1B–E). A neuroradiologist identified the locations that are considered the corticospinal tracts at the levels of the corona radiata, posterior rim of the internal capsule, and cerebral peduncle, and placed the regions of interest bilaterally on FA and ADC maps. The FA and ADC values for each location were compared with those of the normal side. There were no alterations in the FA and ADC values for these areas (Fig 1C–E); color coded maps showed clear depiction of the deep portion of bilateral corticospinal tracts (Fig 1F). These findings indicate no wallerian degeneration associated with prominent dilated PVSs, correlated with the absence of neurologic abnormalities in the patient.

Discussion

Dilated PVSs are round or oval structures with a well-defined, smooth margin that occur along the path of penetrating arteries, are iso-intense relative to CSF, and demonstrate no enhancement after contrast medium administration.¹ Central enhancement representing the blood vessels can occasionally be seen in PVSs. In our case, the multiple foci fulfilled all the MR imaging criteria. Giant PVSs are expanded PVSs that are larger (≥ 1.5 cm) and may have associated focal mass effect,² though giant PVSs were not observed in our case. The precise cause of these PVSs still remains to be elucidated. It is also unknown whether dilated PVSs can affect the neuronal fibers of the white matter. If they affect the neuronal fibers, the degeneration of the neuronal fibers such as wallerian degeneration may be seen on MR imaging.

Wallerian degeneration is the term used to describe secondary degeneration of axons and their myelin sheaths from numerous causes, including infarction, hemorrhage, neo-

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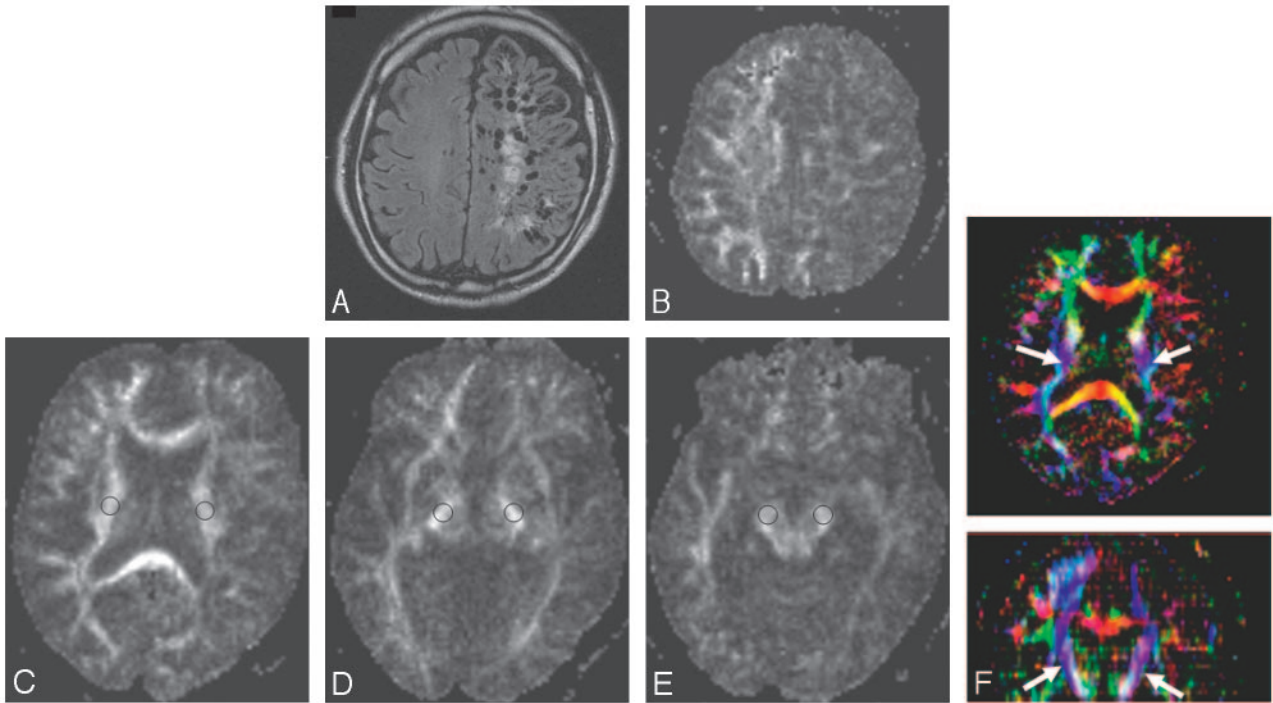


Fig 1. Axial MR images of the brain in a case with multiple, prominent dilated perivascular spaces in left cerebral hemisphere.

A, Fluid-attenuated inversion-recovery (FLAIR) (TR/TE_{eff}/TI, 6000/120/2000; echo-train length, 17) image shows multiple, well-defined, rounded hypointense foci of various sizes in the left cerebral hemisphere with signal intensity similar to that of CSF. Hyperintense areas adjacent to the hypointense foci are also observed in the left cerebral hemisphere. Two small dilated PVs are seen in the right hemisphere.

B, FA map, obtained at a location similar to that in **A** shows decreased FA in the left cerebral white matter. This decreased FA is probably due to partial volume effect of dilated perivascular spaces.

C–E, FA maps at the levels of the corona radiata (**C**), posterior rim of the internal capsule (**D**), and cerebral peduncle (**E**). There was no alteration in the FA and ADC values for the corticospinal tracts of these areas, indicating no wallerian degeneration associated with dilated perivascular spaces. Circles indicate the region of interest we chose. The right and left FA values were 0.60 and 0.59 for the corona radiata, 0.62 and 0.64 for the posterior rim of the internal capsule, and 0.61 and 0.61 for the cerebral peduncle, respectively. The right and left ADC values ($\times 10^{-3}\text{mm}^2/\text{s}$) were 0.76 and 0.76 for the corona radiata, 0.69 and 0.68 for the posterior rim of the internal capsule, and 0.73 and 0.72 for the cerebral peduncle, respectively.

F, Color-coded axial (*upper*) and coronal (*lower*) maps show preservation of the deep portion of the left corticospinal tract (*arrows*). The superficial portion of the left corticospinal tract is not demonstrated probably due to partial volume effect of dilated perivascular spaces.

plasm, and demyelinating disease.^{5–9} Diffusion-weighted imaging and DTI may have utility in the identification of white matter injury corresponding to wallerian degeneration, which is not detectable by conventional MR imaging.^{5–9} There are some reports of chronologic changes on diffusion-weighted images or DTI in wallerian degeneration. Kang et al⁶ reported restriction of water diffusion on diffusion-weighted images 1 day after axonal injury. Castillo and Mukherji⁷ showed high signal intensity on diffusion-weighted images 72 hours after ictus in 20% of cases. There is a report on diffusion tensor imaging in subacute stroke cases, 2–3 weeks after ictus, in which decreased anisotropy has been found,⁸ and another study reported that tensor imaging can delineate wallerian degeneration in chronic infarction cases.⁹

On FA maps, decrease in FA in the periphery of the left cerebral white matter may be due to a partial volume effect of dilated PVs. No alteration in FA and ADC values for the corona radiata, internal capsule, and cerebral peduncle without neurologic findings suggests the absence of wallerian degeneration of the corticospinal tract. Based on these MR imaging findings, multiple, prominent dilated PVs in the cerebral hemisphere caused no secondary damage of neuronal fibers.

It is of interest that fast FLAIR images revealed hyperintense foci in the left hemisphere adjacent to these dilated PVs, which has been described by many authors.^{2–4} Although the

FLAIR finding has not been pathologically confirmed, we speculate that the finding corresponds to chronic ischemic changes, gliosis, or spongiosis associated with dilated PVs. However, ischemic changes that can induce wallerian degeneration may not be the cause because prominent dilated PVs did not cause wallerian degeneration in our patient.

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