Parenchymal abnormalities associated with developmental venous anomalies identified susceptibility-weighted imaging at 3T

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Aims and objectives

Developmental venous anomalies (DVAs), also referred to as venous angiomas or cerebral medullary venous malformations, are the most common vascular malformation of the brain [1-3]. It is generally accepted that they are congenital and follow a benign and clinically asymptomatic course [4]. DVAs are typically diagnosed incidentally during imaging investigations for unrelated symptoms.

Although the parenchyma drained by a DVA is usually normal brain tissue, there have been reports of parenchymal abnormalities in the vicinity of a DVA. The most frequently encountered association is reportedly that of cavernous malformations (CMs). This association is found at a variable rate of 13.3% - 62.3% [2, 5, 6]. On the other hand, high signal intensity abnormalities detected with 1.5 T MRI have been described in 7.8% - 57% of DVAs [5, 8, 9]. In a recent study, Takasugi et al. demonstrated a 54.1% prevalence of associated high signal intensity abnormalities in 61 DVAs evaluated with 3 T MRI [6]. The etiology of the high signal intensity is uncertain, but edema, demyelination or gliosis related to venous stenosis, and altered hemodynamics are possible causes [8].

DVA is a low-flow vascular malformation that is not well imaged with conventional MR sequences. Small DVAs can be easily missed, and therefore contrast-enhanced MRI better enables the detection of DVAs [8]. Reichenbach et al. reported that susceptibility-weighted imaging (SWI) provides unique high-resolution information on vascular lesions such as DVAs and CMs without administration of a contrast medium [10]. Recently, SWI has been used as a complementary imaging modality for identifying DVAs[6]. However, brain parenchymal signal abnormalities associated with DVAs have not been sufficiently investigated with 3 T MRI. In addition, the use of high-resolution 3D sequences of fluid-attenuated inversion-recovery (FLAIR) with 3 T MRI may provide additional information regarding DVAs-associated parenchymal abnormalities, because 3D FLAIR has fewer artifacts from vessels and cerebrospinal fluid pulsation and fewer partial volume effects than 2D FLAIR images and thus may contribute to the detection of subtle high signal intensity lesions [11, 12].

In this study, we evaluated the prevalence of the brain parenchymal signal intensity abnormalities associated with DVAs identified by SWI with 3 T MRI in a large number of patients, and we also assessed the relationship between high signal intensity abnormality and factors such as location, direction, and length of DVAs and hypointense foci (i.e., microhemorrhages or CMs).
Methods and materials

Patients

This study was approved by the ethics committee of our university, and the requirement for written informed consent was waived because of the retrospective nature of this study. Between January 2008 and February 2013, we searched for all MRI reports containing the diagnosis of "venous angiomia" or "developmental venous anomalies". The inclusion criteria in this study were principally based on SWI findings characterized by a cluster of venous radicles that converge into a collecting vein. Cases that were diagnosed as DVAs without the use of SWI images were excluded. Eventually, 168 DVAs in 162 patients (76 men and 86 women; mean age 56.6±18.1 years, range 4-88 years) identified by SWI were included in the study. Clinical indications for each case are summarized in Table 1.

MR sequences

We used three different 3 T MRI machines: the Magnetom Verio (Siemens Medical Solutions, Erlangen, Germany) with a 12-channel phased-array head coil or a 32-channel phased-array head coil; the Achieva (Philips Health Care, Best, The Netherlands) with an 8-channel phased-array head coil or a 32-channel phased-array head coil; and the Ingenia (Philips Health Care, Best, The Netherlands) with a NVC-Base (d Stream) coil. The details of sequence parameters of FLAIR and SWI and the number of DVAs assigned to each machine are shown in Tables 2-4. Other MRI sequences included the following: axial 2D fast spin echo T2-weighted imaging (Verio: TR, 5500-5970 ms; TE, 81-87 ms; echo-train length, 14-18; Achieva: TR, 4005-4500 ms; TE, 90 ms; echo-train length, 13; Ingenia: TR, 4084 ms; TE, 90 ms; echo-train length, 13), axial 3D fast spin echo T1-weighted imaging (Achieva: TR, 300 ms; TE, 16 ms; Ingenia: TR, 300 ms; TE, 16 ms), axial 3D gradient-echo T1-weighted imaging (Verio: TR, 5.46-6.07 ms; TE, 2.42-2.72 ms), axial 3D fast spin echo contrast-enhanced T1-weighted imaging (Achieva: TR, 300 ms; TE, 16 ms; Ingenia: TR, 300 ms; TE, 16 ms), and axial 3D gradient-echo contrast-enhanced T1-weighted imaging (Verio: TR, 6.58-6.79 ms; TE, 3.29-3.39 ms). Contrast-enhanced T1-weighted images were occasionally obtained using a standard dose of contrast medium.

Image analysis

MR images were reviewed by two experienced neuroradiologists who were blinded to the patients' clinical information. Assessments included the presence of signal intensity abnormalities adjacent to DVAs on FLAIR images and the presence of hypointense foci (i.e., microhemorrhages or CMs) around DVAs on SWI. Care was taken not to include signal intensities within the visible vascular structures of DVAs. According to lesion size, high signal lesions were classified into either small (maximum diameter ≤5 mm) or large groups (maximum diameter >5 mm). We also assessed size changes of the high signal lesion with prior and subsequent MRI findings if available. The drainage territory was
defined as the brain parenchyma directly adjacent to the visualized radicles of the DVA. Additionally, we evaluated DVA location (supratentorial or infratentorial), direction of draining vein (deep or superficial), and length of the draining vein. The location of the DVA was classified into supratentorial (cerebral lobe) or infratentorial (pons and cerebellum). The terminal or draining vein to which the caput medusae joined was classified as either a deep (toward the ventricle) or superficial (toward the brain surface) draining vein. Concerning the length of the draining vein, the draining vein was long if the depth was juxtacortical and the direction was deep or if the depth was periventricular and the direction was superficial. The draining vein was short if the depth was juxtacortical and the direction was superficial or if the depth was periventricular and the direction was deep. All other DVAs were categorized as a median draining vein. Imaging findings were determined by consensus between the two neuroradiologists.

The degree of underlying white matter disease was assessed and classified as none, minimal (£15 foci of abnormal hyperintensity), mild (16-35 foci), or severe (£36 foci or confluent abnormal signal intensity).

**Statistical methods**

The relationships between high signal lesions adjacent to DVAs and age, gender, location, direction, length of draining vein, and presence of hypointense foci were assessed. Logistical regression models were used to assess the association of signal intensity change with the factors either included (all cases) or excluded (a subgroup of none or minimum white matter disease) in the models. All other numeric variables were summarized by mean and range and compared using a $c^2$ test. P-values <0.05 were considered statistically significant. Statistical analysis was performed using commercially available software (SPSS 21.0, Chicago, IL, USA).
Table 1: Clinical Indications for MR examinations in patients with DVAs Note: Total n = 162

<table>
<thead>
<tr>
<th>Clinical Indication</th>
<th>Patients</th>
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<tbody>
<tr>
<td>Cerebral vascular disorder</td>
<td>64</td>
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<tr>
<td>Brain tumor</td>
<td></td>
</tr>
<tr>
<td>Primary brain tumor</td>
<td>22</td>
</tr>
<tr>
<td>Brain metastasis</td>
<td>22</td>
</tr>
<tr>
<td>Degenerative disease</td>
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</tr>
<tr>
<td>Dementia</td>
<td>9</td>
</tr>
<tr>
<td>Others</td>
<td>2</td>
</tr>
<tr>
<td>Medical checkup of the brain</td>
<td>10</td>
</tr>
<tr>
<td>Trauma</td>
<td>7</td>
</tr>
<tr>
<td>Mental disorder</td>
<td>7</td>
</tr>
<tr>
<td>Brain aneurysm</td>
<td>5</td>
</tr>
<tr>
<td>Head and neck tumor</td>
<td>4</td>
</tr>
<tr>
<td>Demyelinating disease</td>
<td>3</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>2</td>
</tr>
<tr>
<td>Others</td>
<td>5</td>
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</tbody>
</table>

Table 2: SWI sequences parameters used in the imaging protocol ch: channel, FOV: Field of View, Acq: Acquisition, MPS: Matrix Phase Slice, SENSE: Sensitivity Encoding, TR: Repetition Time, TE: Echo Time, FA: Flip Angle

<table>
<thead>
<tr>
<th></th>
<th>Siemens Verio 3T</th>
<th>Philips Achieva 3T</th>
<th>Philips Ingenia 3T</th>
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<tbody>
<tr>
<td>Coi</td>
<td>12ch</td>
<td>32 ch</td>
<td>8ch</td>
</tr>
<tr>
<td>FOV (mm)</td>
<td>210</td>
<td>210</td>
<td>230</td>
</tr>
<tr>
<td>Matrix Acq voxel MPS (mm)</td>
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<td>0.80/0.60/0.80</td>
<td>0.72/0.72/1.60</td>
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<td>3.0/1.0</td>
<td>2.0/1.0</td>
</tr>
<tr>
<td>TR/TE/FA (ms/ms/deg)</td>
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<td>25/20/16</td>
<td>21/32/20</td>
</tr>
<tr>
<td>Total scan time (min:sec)</td>
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<td>4:15</td>
<td>4:13</td>
</tr>
</tbody>
</table>

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Table 3: FLAIR sequences parameters used in the imaging protocol ch: channel, FOV: Field of View, TI: Inversion Time, Acq: Acquisition, MPS: Matrix Phase Slice, SENSE: Sensitivity Encoding, NA: Not Applicable, TR: Repetition Time, TE: Echo Time, FA: Flip Angle

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<table>
<thead>
<tr>
<th></th>
<th>Siemens Vario 3T</th>
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<th>Philips Achieva 3T</th>
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<th>Philips Ingenia 3T</th>
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<tbody>
<tr>
<td><strong>Coil</strong></td>
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<td>32ch</td>
<td>8ch</td>
<td>32ch</td>
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<td>3D</td>
<td>2D</td>
<td>3D</td>
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<td>3D</td>
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<tr>
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<td>200</td>
<td>235</td>
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<td>TI (ms)</td>
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<td>1800</td>
<td>2556.6</td>
<td>2550</td>
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<td>Matrix Acq voxel MPS (mm)</td>
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<td>1.0×1.0×1.0</td>
<td>0.6×0.6×3.0</td>
<td>0.9×0.9×0.8</td>
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<tr>
<td>SENSE factor (Phase/Slice)</td>
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<td>2.0/NA</td>
<td>3.0/NA</td>
<td>3.0/NA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TR/TE/FA (ms/ms/deg)</td>
<td>12000/82/90</td>
<td>5000/550/90</td>
<td>10000/84/90</td>
<td>10000/625/90</td>
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<tr>
<td>Total scan time (min/sec)</td>
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<td>3.20</td>
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</table>

Table 4: Number of DVAs assigned to each MRI machine ch: channel, No.: Number

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Results

Fifty-one of 168 DVAs (30.4%) were also examined with contrast-enhanced T1-weighted images, in all of which DVAs were also detectable on contrast-enhanced T1-weighted images.

Of the 168 DVAs identified, 57 had associated high signal abnormalities in the drainage territory (33.9%) (Table 5). One of 57 DVAs with high signal abnormalities was excluded because of demyelinating lesions in multiple sclerosis overlapping the drainage abnormalities. Of the remaining DVAs imaged in this study, 56/167 (33.5%) DVAs had adjacent high signal abnormalities (Table 5). An adjusted prevalence rate of 18/81 (22.2%) was obtained by including only patients with none or minimum underlying white matter disease. Twenty-six DVAs were determined as associated hypointense foci (i.e., microhemorrhages or CMs) (26/168 DVAs: 15.5%).

Table 6 outlines the association of high signal abnormalities with other factors using a total of 167 DVAs from 161 patients. The location of DVAs was associated with DVA-related high signal abnormalities in all cases. In particular, high signal lesions were more likely to be associated with supratentorial DVAs than infratentorial DVAs (p<0.05). After adjusting for underlying white matter disease, none of the DVAs were identified in infratentorial areas. The direction into the deep venous system of DVAs was associated with DVA-related high signal abnormalities (p<0.05). This achieved statistical significance, even after adjusting for underlying white matter disease. In particular, high signal abnormalities were more likely to be associated with the deep direction than the superficial direction in all cases. Underlying white matter disease was associated with DVA-related high signal intensity abnormality (p<0.05). In particular, high signal lesions were more likely to be associated with severe white matter disease than other categories (p<0.05). Age was also associated with DVA-related high signal intensity abnormality (p<0.05). This did not remain significant after adjusting for underlying white matter disease. Other factors (hypointense foci, length of draining vein, and gender) were found to have no association with high signal lesions. In a subgroup, sample size was too small to attain the sufficient statistical power regarding the location and the length of draining veins.

Eleven of 18 DVAs (61.1%) showed a large, classic high signal intensity abnormality with major axis >5 mm surrounding the DVAs and extending into the white matter (large group) (Fig. 1), while 7 of 18 DVAs (38.9%) showed a small high signal intensity abnormality with major axis ≤5 mm surrounding the DVAs (small group) (Fig. 2). High signal lesions in the small group were all evaluated using 3D FLAIR. In the large group, the mean maximum diameter of the high signal lesion was 14.5 ± 7.4 (6-25 mm), and the patients' mean age was 58.9 ± 14.3 years (28-72 years). In the small group, the mean maximum diameter of the high signal lesion was 3.0 ± 1.2 mm (2-5 mm), and the patients' mean age was 30.1 ± 11.8 years (9-41 years). Patients in the small group were significantly younger than those in the large group (p<0.05).
Prior and subsequent MRI findings were available in 77/167 DVAs (46.1%). Among them, two DVAs had increased in size during the follow-up period (8.4 years and 11.7 years of follow-up, respectively) (Fig. 3): a 67-year-old man and a 77-year-old woman with no symptoms. The remaining 75 cases showed no change in size during the follow-up period (0.08-13.3 years of follow-up).
Table 5: Summary of characteristics of three groups of patients based on high signal intensity abnormalities in DVA territory Note: White matter disease indicates degree of overall white matter high signal lesions as outlined in the Results section. #One case with very extensive additional non-DVA-associated white matter high signal intensity abnormality was excluded from analysis. †Values in cells are mean and standard deviation (range). No.: Number

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Table 6: Associations between high signal intensity abnormality within DVA drainage region and other factors Note: Subgroup includes patients with none or minimum white matter disease. DF indicates degree of freedom. §In all cases, location, direction, white matter disease and age of DVAs was associated with DVA-related high signal intensity abnormalities using a logistic regression model. In a subgroup, direction of DVAs was associated with DVA-related high signal intensity abnormalities (p<0.05). †Significant OR
with p<0.05 using a logistic regression model. ‡Values in cells are mean and standard deviation.

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Fig. 1: A 69-year-old male with DVA in the left parietal lobe. a. SWI shows DVA in the left parietal lobe (arrow). b. 2D FLAIR shows a large, classic high signal intensity abnormality surrounding the DVA, extending into the white matter (arrow).

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Fig. 2: A 39-year-old female with DVA in left frontal lobe. a. SWI shows a low signal intensity structure of the DVA (arrow). b. 3D FLAIR shows a small high signal intensity abnormality adjacent to the DVA (arrow).

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Fig. 3: A 67-year-old man with DVA in the left frontal lobe. a. 2D FLAIR image shows a large high signal intensity abnormality in the left frontal lobe (arrow). b. Follow-up 2D FLAIR image shows a high signal lesion increases in size during the 5 years interval. c. Follow-up 2D FLAIR image 8.4 years later shows that the high signal lesion is stable in size. d. SWI shows a low-signal-intensity structure of the DVA (arrows). e. Contrast-enhanced T1-weighted image shows DVA is enhanced by contrast agent administration (arrow).

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Conclusion

Discussion

SWI combines the detailed spatial resolution of 3D gradient-echo imaging with the ability to detect blood with a reduced oxygenation level. This unique description of anatomical and physiological features makes it possible to investigate slow-flow vascular malformations such as DVAs and CMs as well as small hemorrhages [10]. On the other hand, other non-contrast MR sequences including spin-echo sequences, time-of-flight MR angiography or MR venography, and 2D T2*-gradient echo techniques are suboptimal for malformed vessels, which exhibit slow multidirectional flow [13]. Thus, SWI appears suitable for identifying DVAs and related CMs or small hemorrhages.

This study focused on parenchymal abnormalities in the drainage territory of DVAs identified by SWI. Four studies, including ours, have demonstrated the prevalence of high signal intensity adjacent to DVAs [5, 6, 8]. Based on the three previous studies, the prevalence of high signal intensity abnormalities in the drainage territory of DVAs was variable, ranging from 7.8% - 57%. In our study, high signal intensity alterations accounted for 33.5% of all cases and 22.2% in a subgroup of patients with none or minimum underlying white matter disease, excluding patients with moderate to severe white matter disease. Compared to the three previous studies of high signal lesions associated with DVAs, our study consisted of the second largest number of DVAs (n=168), and was not much smaller than the largest number of DVAs (n=175), which was reported by Santucci et al. [8]. The remaining two studies had relatively small numbers of DVA cases identified by MRI (n=60 and 61, respectively) [5, 6]. Moreover, in these two studies, no specific mention was made of exclusion criteria related to potential signal intensity changes associated with adjacent non-related pathology [5], and no attempt was made to control for more widespread white matter signal intensity alterations common in adult patients [5, 6]. MRI methods to identify DVAs were variable, including contrast-enhanced MRI in two previous studies [5, 8] and SWI in the present study and one previous study [6]. Therefore, variation of the prevalence may be due to varying inclusion and exclusion criteria, the number of patients included, and MRI methods. Nonetheless, the high prevalence of associated abnormal high signal lesions should be noted, as the use of SWI is expected to bring more opportunities for identifying DVAs than non-contrast conventional MRI sequences on the basis of everyday clinical experience.

In the present study, high signal lesions adjacent to DVAs were evaluated with 2D FLAIR or 3D FLAIR, and 3D FLAIR was predominantly used in 131 out of 168 DVAs (78.0%). Previous investigators have used 2D FLAIR or 2D T2-weighted images in the evaluation of high signal lesions [5, 6, 8], but there have been no reports on the use of 3D FLAIR in the detection of high signal lesions associated with DVAs. Kakeda et al. reported that with regard to the conspicuity and detection of most brain lesions, 3D FLAIR was equal or superior to 2D FLAIR, and for these lesions the mean contrast ratios were higher on 3D
FLAIR than on 2D FLAIR images using 3 T [14]. In addition, we used thin slice 3D FLAIR images (1-3 mm) in the evaluation of high signal lesions adjacent to DVAs, leading to more precise evaluation and detection of small high signal lesions. Small lesions (e.g., ≤5 mm in diameter) might be missed if interpreted by 2D FLAIR with a 5-mm slice and 1-mm gap. For these reasons, we speculate that we could identify small high signal lesions associated with DVAs owing to the use of high-resolution 3D FLAIR in the current study.

The etiology of high signal intensity lesions remains uncertain. The pathology of the lesions also remains unknown, but some possibilities include gliosis, demyelination or edema due to chronic mild venous hypertension caused by anomalous venous drainage [15]. Santucci et al. reported that DVAs with high signal lesions were more frequently seen in older patients compared to patients without high signal lesions [8], and our results support their conclusion. In addition, our results showed that patients with small high signal lesions (small group) were significantly younger than those with large and classic high signal lesions (large group). We infer that high signal lesions adjacent to DVAs may increase in size with age. Two cases that had been followed on MRI showed increases in the size of high signal lesions, although follow-up was limited to about 13 years in the present study. Leukoaraiosis is an age-related neurodegenerative condition that appears as an area of hyperintense signal in the white matter on MRI, resulting from white matter ischemia. It is characterized histologically by demyelination, loss of glial cells, and vacuolization (spongiosis) [16]. Moody et al. reported that there may be a mechanistic link between leukoaraiosis and collagenous thickening of venous walls, rather than an incidental association, with both pathologies independently occurring near the ventricle [17]. Increased resistance to venous blood flow, resulting from the venous stenosis, might induce chronic ischemia and/or edema in the deep white matter, leading to leukoaraiosis [18]. DVAs might have a similar pathophysiology with aging and might occur with high signal lesions. Thus, altered hemodynamics and susceptibility to ischemia due to aging may be one reason why DVAs in older patients accompany high signal lesions more frequently and the large size of high signal lesions is more frequently seen in older patients. We also infer that a small high signal lesion might be an early finding of a large and classic high signal lesion related to DVAs.

We used SWI at 3 T to detect hypointense foci, indicating microhemorrhage or CMs. SWI is quite sensitive to the presence of even small amounts of hemorrhage. In addition, 3 T MRI shows the susceptibility effect greatly, resulting in the detection of hypointense foci more easily. However, the prevalence of hypointense foci was 15.5% in the current study, indicating almost the same level (up to 18%) of previous reports using conventional MRI methods such as 2D gradient echo at 1.5T [8, 19]. On the other hand, Takasugi et al. reported a higher prevalence (62.3%) of hypointense foci than the present study, although they also used SWI at 3 T [6]. The reason for this discrepancy in the prevalence of microhemorrhage and CMs between our study and Takasugi’s study is unknown. They also reported that hypointense foci showed a significant association with high signal abnormalities related to DVAs on T2-weighted images. However, our results showed no significant association between hypointense foci and high signal abnormalities related to
DVAs. Further studies in a larger number of cases using SWI will be necessary to clarify the association between hypointense foci and high signal abnormalities related to DVAs.

This study has several limitations. First, although our study population was as large as that studied by Santucci et al., and larger than that in other studies [5, 6, 8], prospective review of more cases may be necessary to confirm the prevalence of signal intensity abnormalities associated with DVAs using SWI at 3 T. Moreover, our study included 2D FLAIR as well as 3D FLAIR to evaluate hyperintensity abnormalities associated with DVAs, as 3D FLAIR was not necessarily used in all cases. However, high-resolution 3D FLAIR is mandatory to specifically identify small high signal lesions related to DVAs. Therefore, further studies using 3D FLAIR at 3 T are also required to validate the present results. Second, we hypothesized that SWI may be almost equal to contrast-enhanced T1-weighted images in the detection of DVAs, but this is not clear. The detectability of DVAs should be studied between SWI and contrast-enhanced MRI using 3 T. Third, we did not sufficiently obtain the prior and subsequent MR examinations to detect any interval change of high signal lesions related to DVAs for the current cases. This will be necessary to observe interval changes of high signal lesions related to DVAs over longer time periods.

**In conclusion,** the prevalence of hyperintense lesions associated with identified DVAs was 33.5%, and was 22.2% in a subgroup that included only patients with none or minimum underlying white matter disease. The prevalence of associated hypointense foci was 15.6%. The high prevalence of associated abnormal high signal lesions should be noted, as the use of SWI is expected to bring more opportunities for identifying DVAs on the basis of everyday clinical experience. Our results suggest that the size of associated high signal intensity abnormalities may increase with aging.
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