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Low-signal-intensity rim on susceptibility-weighted imaging is not a specific finding to progressive multifocal leukoencephalopathy

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ABSTRACT

Background: Low-signal-intensity (LSI) rim along deep layers of the cerebral cortex is reportedly a susceptibility-weighted imaging (SWI) finding in progressive multifocal leukoencephalopathy (PML). We aimed to evaluate whether this finding can be identified in diseases other than PML.
Methods: We retrospectively reviewed brain MR images from 5605 patients who underwent SWI at 3T; 370 patients with various diseases, who showed cortical and subcortical FLAIR high-signal lesions including U-fiber, were enrolled. The presence or absence of LSI rim on thin-slice SWI and hyperintense cortical signal (HCS) on T1-weighted images adjacent to LSI rim was analyzed. Signal changes of the LSI rim were assessed on serial SWI, if available.
Results: Twenty-five of the 370 patients (6.8%) showed SWI LSI rim, in infarct ($n = 22$) and encephalitis ($n = 3$). HCS was apparent adjacent to SWI LSI rim in 17 patients (15 infarct, 2 encephalitis). Serial SWI was available for 17 patients, of whom 10 patients (8 infarct, 2 encephalitis) presented LSI rim later than 45 days after onset.
Conclusion: LSI rim can be observed in infarct and encephalitis. Therefore, this finding is not specific to PML. LSI rim appears to be associated with HCS.

1. Introduction

Susceptibility-weighted imaging (SWI) is a magnetic resonance (MR) imaging technique used in several clinical applications [1–3]. SWI utilizes susceptibility differences between adjacent tissues to produce tissue contrast and is exquisitely sensitive to the detection of diamagnetic and paramagnetic substances, such as blood products, iron, and calcium. Since its introduction, SWI has offered additional diagnostic information in a wide spectrum of brain diseases [1–3]. Recently, we reported a new finding in progressive multifocal leukoencephalopathy (PML) that is a low-signal-intensity (LSI) rim along deep layers of the cerebral cortex identified by SWI in PML lesions [4]. More recently, this finding was followed by several investigators [5,6]. We suggested that the LSI rim along deep layers of the cerebral cortex is of clinical value for early diagnosis of PML. However, it remains unclear whether the LSI rim is specific to PML. The purpose of this study was to retrospectively evaluate whether the LSI rim along deep layers of the cerebral cortex can be identified in diseases other than PML that involve the subcortical and cortical regions.

2. Materials and methods

2.1. 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 study design. We reviewed radiological records of 5605 patients who underwent brain SWI at 3T in our institution from January 2008 to December 2014. We excluded PML cases in this study. Among these cases, fluid-attenuated inversion recovery (FLAIR) images were further investigated with regard to the presence of cortical and subcortical high-signal lesions involving U-fiber. Among them, 378 patients with cortical and subcortical FLAIR high-signal lesions were included. However, 8 of the 378 patients were excluded because the MR image quality was insufficient for evaluation because of motion or susceptibility artifacts. Finally, 370 patients (233 males and 137 females; mean age, 62.8 ± 19.0 years) with cortical and subcortical FLAIR high-signal lesions were enrolled in this study. Details of the clinical diagnoses of the 370 patients are summarized in Table 1.

2.2. MR sequences

Images were obtained using the following two different 3-T MR scanners: the Achieva (Philips Health Care, Best, The Netherlands),

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Table 1
Clinical diagnoses of all patients with cortical and subcortical FLAIR high-signal lesions including U-fiber.

Clinical diagnosis		Patients ($n = 370$)
Cerebral vascular disorder		203
Brain tumor	Primary brain tumor	48
	Brain metastasis	55
Trauma		26
Encephalitis		14
Encephalopathy		7
Meningitis		4
Demyelinating disease		4
Brain malformation		4
Degenerative disease		3
Abscess		2

with a 32-channel phased-array head coil and the Ingenia (Philips Health Care, Best, The Netherlands), with a NVC-Base (d Stream) coil. Routine MR sequences such as T1-weighted, T2-weighted, FLAIR, and diffusion-weighted imaging (DWI) were achieved in all patients, in addition to SWI. The Achieva and Ingenia scanners performed SWI using the following parameters: TR, 21–25 ms; TE, 32–37 ms; flip angle, 20°; FOV, 25 cm; matrix size, 512 × 320; section thickness, 0.8 mm; and acquisition time, 4 min 13 s–5 min 12 s. To achieve optimal diagnostic quality image, SWI was displayed in the transverse plane using the slice thickness of 1.6 mm. The Achieva and Ingenia scanners performed 3D FLAIR sequence using the following parameters: TR, 6000 ms; TE, 310 ms; TI, 2000 ms; turbo factor, 203; SENSE factor, 3; FOV, 25 cm; matrix size, 480 × 256; section thickness, 1.14 mm; and acquisition time, 5 min 30 s.

2.3. Image analysis

Signal intensities of cortical and subcortical lesions in various sequences and topographic distribution of the abnormalities and their extent were independently evaluated by two experienced neuroradiologists (M.U. and M.M.). Whether LSI rim was present along deep layers of the cerebral cortex adjacent to cortical and subcortical FLAIR lesions, including U-fiber on thin-slice SWI images (1.6 mm), was determined in comparison with normal-appearing deep layers of the cerebral cortex in the same slices independently. Discordant findings on imaging were resolved by consensus between the two neuroradiologists. The LSI rim along deep layers of the cerebral cortex on thin-slice SWI images was evaluated each time if the same patients had multiple sequential MR examinations. Other morphological types of LSI, such as dot-like or cluster suggesting hemorrhage, were also evaluated on SWI images. We carefully reviewed the configurations of SWI LSI to ensure hemorrhage.

In the preliminary review of several cases with LSI rim, we noticed that T1 shortening of the cortex adjacent to LSI rim was frequently present. Therefore, parenchymal abnormalities adjacent to the SWI LSI rim were also assessed with respect to the presence or absence of hyperintense cortical signal (HCS) on T1-weighted images, suggesting cortical laminar necrosis.

3. Results

Twenty-five of the 370 patients (6.8%) showed LSI rim on thin-slice SWI images. These patients included 15 males and 10 females (mean age: 63 years; range: 35–84 years). Clinical diagnoses of these 25 cases were infarct ($n = 22$) and encephalitis ($n = 3$; 1 herpes simplex virus and 2 undetermined pathogens). A summary of clinical and MRI findings is shown in Table 2. Representative cases were shown in Figs. 1 and 2. With regard to HCS on T1-weighted images, 17 (15 infarct, 2 encephalitis) of the 25 cases (68%) showed HCS adjacent to the SWI LSI rim (Table 2). The regions of HCS were more extensive than those of LSI rim in 5 cases (Fig. 1), while the regions of LSI rim were more extensive than those of HCS in 5 cases. The regions of HCS and LSI rim were almost equal in 7 cases (Fig. 2). Six patients with infarct showed dot or a cluster type of LSI on SWI within FLAIR hyperintensity lesions. These types of SWI LSI were thought to represent hemorrhage within the infarcted lesions.

Serial MR examinations were conducted in 17 cases. The time from the onset to follow-up MR examinations ranged from 1 to 2481 days (mean 416 ± 539 days). Ten of the 17 cases (58.8%) showed SWI signal changes during the investigated period (Figs. 1 and 2). Clinical details and imaging findings of these 10 patients are summarized in Table 3. Signal changes were observed 45–1448 days (mean 390 ± 419) after onset. LSI rim persisted in 17 cases during the follow-up periods (maximum 2481 days after onset). Based on these results, the LSI rim appeared at least 45 days after onset, and HCS on T1-weighted images was also apparent at the same time in 7 of the 10 cases (Figs. 1 and 2).

4. Discussion

In our previous report [4], we suggested that the SWI LSI rim may be of clinical importance for the early diagnosis of PML. However, it was uncertain whether this finding could be specific to PML. Based on the current study, we have shown that LSI rim along deep layers of the cerebral cortex is apparent in patients with infarct and encephalitis. Therefore, this finding is not specific to PML.

The SWI appearance of our cases is peculiar, demonstrating LSI rim along the deep cortical layers. The LSI rim appears to be located in deep layers of the cerebral cortex, with or without U-fiber, and not in superficial layers of the cortex. To the best of our knowledge, this finding, apart from PML, was barely reported in one of the infarct cases in a pictorial review by Tsui et al. [2], featuring SWI for the differential diagnosis of cerebral vascular pathology. Kesavadas et al. reported that SWI detected diffuse linear hypointensities along the gyral margins in two cases with hypoxic ischemia [7]. Although the SWI findings in these two cases are similar to those in our cases, the LSI rim in their cases was very thick and appeared to be located in all layers of the cortex, with or without U-fiber. We used thin-slice SWI images (1.6-mm slice thickness) to more precisely evaluate lesion topography in this study and in a previous report [4]. On the other hand, Kesavadas et al. used 9- to 12-mm thick, minimum-intensity projection slabs for SWI images [7]. We inferred that this may have caused misinterpretation of localization because of partial volume averaging of a lesion as a focus of susceptibility. In our PML cases previously reported [4], using minimum-intensity projection slabs for SWI images, the LSI rim appeared to be located in the entire cortex, although the LSI rim was identified along the deep layers of the cortex using thin-slice SWI images. Therefore, hypoxic encephalopathy cases as well as infarct cases may cause LSI rim along deep layers of the cortex, which is similar to the finding seen in PML cases. However, we have experienced no cases of hypoxic encephalopathy. Further investigation is necessary to confirm the topography of LSI rim in cases with hypoxic encephalopathy using thin-slice SWI images.

HCS on T1-weighted images has been described in several central nervous system conditions, most notably in hypoxia–ischemia, status epileptics, hypoglycemia, and mitochondrial disorders [8]. This finding is commonly thought to be a radiologic marker of cortical laminar necrosis, which is a process of neuronal loss and death with resultant gliosis. In our study, it should be noted that 68% of cases with the LSI rim concomitantly showed HCS on T1-weighted images adjacent to the LSI rim. It was reported that all three cases by Tsui et al. and Kesavadas et al. showed the presence of HCS adjacent to the LSI rim [2,7]. Recently, it was reported that 61.2% of PML cases had HCS adjacent to subcortical PML lesions [9]. In one of the two PML cases described in our previous report [4], HCS was present adjacent to PML lesions, and the LSI rim was observed adjacent to HCS. Consequently, the LSI rim appears to be associated with HCS, regardless of the spectrum of diseases. In MR findings time course of nine cases with LSI rim and HCS (Table 3),