Diversity of Cortical and Subcortical MS Pathology/Lesions Revealed by 7T MRI

A. N. Dula^{1,2}, S. Pawate³, E. B. Welch^{1,4}, R. G. Avison¹, S. Sriram³, J. L. Creasy², J. C. Gore^{1,2}, and M. J. Avison^{1,2}

¹Institute of Imaging Science, Vanderbilt University, Nashville, TN, United States, ²Radiology and Radiological Sciences, Vanderbilt Medical Center, Nashville, TN, United States, ³Neurology and Immunology, Vanderbilt Medical Center, Nashville, TN, United States, ⁴3MR Clinical Science, Philips Healthcare, Cleveland, OH, United States

Purpose

MRI is well established as the imaging modality of choice for the diagnosis of MS and its response to therapy. The improvements in sensitivity and spatial resolution that have accrued with increasing field strengths, up to the current clinical standard of 3T, have provided corresponding improvements in the conspicuity of MS lesions [2-4], and with the recent introduction of 7T human clinical scanners, there is reason to expect further improvement in sensitivity, as well as novel information regarding MS pathology in vivo [5, 6]. While the classical view of MS is one of inflammatory

demyelination regarding MS pathology in Vivo [5, 6]. While the classical demyelinating lesions in white matter, cortical grey matter (GM) atrophy and focal cortical lesions are also hallmarks of the disease [7, 8]. Cortical lesions show evidence of non-inflammatory demyelination [9, 10], and there is evidence that GM pathology may contribute significantly to the clinical disability in patients [11]. Similarly, there is strong neuropathological evidence that WM lesions originate from perivascular inflammatory foci [6], suggesting that these small lesions may represent early biomarkers of relapse and/or response to therapy. Both GM and small perivascular WM lesions are poorly visualized by standard MRI protocols at 1.5 or 3T. The goal of the present study was to assess the potential for 7T neuroimaging of MS, with particular emphasis on improving conspicuity of MS pathology not previously observed by MRI at lower fields.

Methods

Using a protocol approved by the Vanderbilt University Medical Center IRB, patients with progressive MS were scanned on a Philips Achieva 7T scanner (Philips Healthcare, Cleveland, OH) equipped with a 16-channel NOVA head coil. The 7T MS protocol included: FLAIR (TR/TI/TE = 15000/2800/44ms; 0.6x0.6x2.5mm voxel; imaging time = 6:30 min); T1W 3D fast field echo (FFE, TR/TE = 9.1/4.5 ms; FA = 10°; 0.72x0.72x1.75 mm voxel; imaging time = 9 min); Multi-echo gradient echo (TR/TE1/deltaTE = 22/12/2ms FA = 10°; 0.72x0.72x0.9 mm voxel; imaging time = 4:04 min); Pre- and post-contrast T1W spin echo (TR/TE = 4.3/2.24ms 0.5x0.5x3mm voxel; imaging time = 3:47 min). For the contrast-enhanced exams, images were collected following ½ (0.05 mg/kg) and full (0.1 mg/kg) dose Gd-DTPA (Magnevist®, Schering, Berlin, Germany). Signal

intensities (SI) of regions of interest (ROIs) were measured on normalized T1W images. Contrast to noise ratios (CNR) were examined for varying doses of Gd-DTPA.

Results

Cortical GM pathology was evident as focal hypointensities on high-resolution 3D-FFE images (Fig 1). These lesions were not evident on FLAIR, nor on T1W SE pre- and post-contrast, consistent with previous studies at lower fields. High resolution T2*W and susceptibility weighted (SW) images revealed perivascular lesions that appear to form cuffs around deep medullary veins (Fig 2). Finally, full dose contrast yielded ~2-fold greater enhancement in lesions than did ½ dose (Fig 3),





Figure 1 – 7 Tesla images of progressive MS patient using A) FLAIR and B) T1-weighted 3D FFE. The cortical lesions are not visible using FLAIR, but are hypointense on T1-weighted images as indicated by arrows.

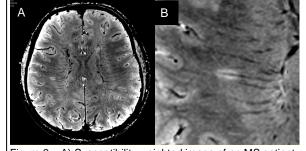


Figure 2 – A) Susceptibility weighted image of an MS patient acquired at 7T, B) Magnified area of lesion demonstrating perivascular lesion location.

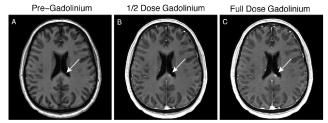


Figure 3 – Images acquired at 7T on MS patient A) pre-contrast, B) after administration of ½ dose of contrast agent, and C) after full dose of Gd-DTPA contrast agent.

suggesting that with the increased SNR available at 7T, lower contrast doses may provide lesion detectability comparable to standard dose at 1.5 or 3T.

Conclusions

The course of MS is highly variable and better markers of disease activity and response to therapy are needed. In addition to the pathology revealed by MRI at 1.5 and 3T, ultra-high field MRI also reveals cortical and perivascular MS lesions that reflect different aspects of MS neuropathology.

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