

## Cortical Lesions in MS: Assessment at 7T

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**Introduction** Multiple sclerosis is generally thought of as a white matter (WM) disease [1], yet there is limited correlation between white matter lesion (WML) load and neurological deficits found clinically [2]. Cortical damage was identified early [3,4] and has recently been correlated with the neurological episodes of MS patients [5]. Pathologically, cortical lesions (CLs) are classified into four types: leukocortical, (I), intracortical (II), subpial (III), and lesions that encompass all six layers of the cortex (IV). Histologically, subpial (III) lesions are the most numerous totaling 60-75% of all lesions, followed by types II, I, IV at 16-17%, 4-15%, and 4-8%, respectively. CL imaging at standard field strengths has had limited success [8], but recent studies at 7T using 2D T2\* weighted imaging look promising [9,10,11]. The objective of this study was to evaluate the potential of 7T high resolution 3D imaging using a white matter attenuated turbo field echo (WHAT-TFE) and 3D T2\*/susceptibility weighted magnitude and phase imaging (SWI) for detecting CLs.

**Materials and Methods** With IRB approval, seven patients (4 RRMS and 3 progressive MS) and healthy controls were scanned with a 7.0 Tesla MRI scanner using a 16 channel receive coil. The WHAT-TFE sequence is an inversion recovery magnetization preparation sequence with TI selected to null the WM signal (TI = 550ms, shot interval TS = 3700ms, TR/TE/flip angle = 4.1/1.6/8°, voxel size 0.4x0.4x0.7 mm<sup>3</sup>). SW images were acquired using a non-spoiled gradient echo sequence with TR/TE/flip angle = 23/12/2-8°, voxel size 0.23x0.23x0.7 mm<sup>3</sup>). Phase images were calculated from complex raw data using high-pass filtering. For comparison, 5 patients had pre-, and 2 patients had pre- and post-contrast T1-weighted IR-TFE scans (TI/TS = 4000/2000ms, 0.23x0.23x1.4 mm<sup>3</sup>) for assessment of lesion enhancement. CLs were marked by 3 readers; two were previously trained for cortical lesion detection [12].

**Results** WHAT showed excellent GM/WM and WM/WML contrast. CLs were hyperintense compared to normal-appearing cortical gray matter (NACGM). Lesions were also brighter than NACGM on SWI magnitude images, but with diminished contrast. CLs were dark in T1-w IR-TFE (Fig. 1). In the post-contrast IR-TFE images, small enhancing veins were visualized adjacent to non-enhancing lesions. Localized *in vivo* cortical contrast changes that were identified as lesions were confirmed by specimen MR and histological comparisons (Fig. 2). Results of lesion counting are listed in Table 1. Regions with contrast inconsistency due to RF inhomogeneity were excluded from the cortical lesion assessment. Prior specimen training did not appear to affect significantly the ability to identify cortical lesions, and the absolute numbers were similar for the two readers who counted all cases. Reader DP counted only 5/7 cases, although the percentages of the lesions found are similar to the other two readers. Fewer lesions were seen in SWI overall due to lowered image

contrast. Type I lesions are the most easily identified and comprised 70-80% of all counted lesions, and the proportion of Types II-IV were similar in WHAT and SWI.

**Discussion** Cortical lesions were best seen with WHAT, being hyperintense to NACGM. Dark lesions in IR-TFE were much harder to detect. Contrast in SW images was lower than reported in prior studies [9,10,11] due to patient motion, lower SNR and the increased sensitivity to RF inhomogeneity of 3D scans. There was little reader variability even with prior training, but "consensus" reads are needed confirm whether the readers identified the same or different lesions.

**References** [1] McDonald, W.L.; et al. Ann. of Neur. 50:121-127; 2001. [2] Barkhof, F. Curr. Op. in Neur. 15:239-245; 2002. [3] Sander, M. Monat. der Psych. und Neur. IV:429-436; 1898. [4] Taylor, E.W. Deut. Zeit. für Nerv. 5:1-26; 1892. [5] Rovaris, M.; et al. Am. Jour. of Neurorad. 21:402-408; 2000. [6] Geurts J Neurol 2008, [7] Bo 2003, [8] Geurts Radiology 2005, [9] Kollia, AJNR 2009, [10] Metcalf AJNR 2008, [11] Mainero, Neurology 2009, Schmalbrock, ISMRM 2008,3225, Pitt Arch Neurol 2010

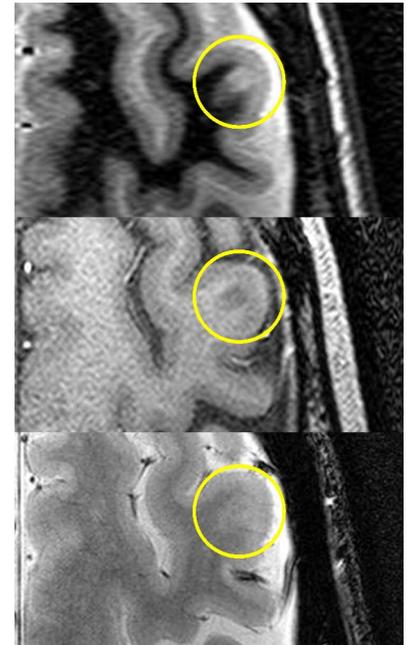


Figure 1: Cortical lesion contrast changes with the sequence used. (a) WHAT, (b) pre-contrast IR-TFE, and (c) second echo SWI.

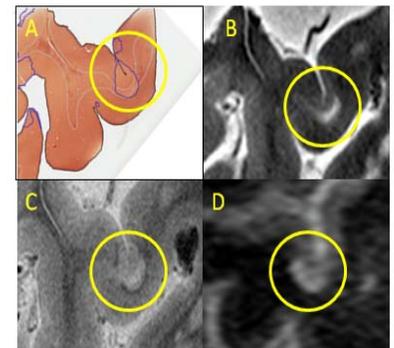


Figure 2: Specimen and histological comparisons can confirm that *in vivo* contrast differences are indicative of cortical lesions. (A) histological section, (B) WHAT, (C) SWI, (D) WHAT *in vivo*.

Table 1: Cortical lesion counts for WHAT and SWI. Reader DP only counted 5/7 cases. Fewer lesions were seen in SWI despite the higher resolution due to low contrast.

| Reader | Sequence | Mixed (I, I-III, I-IV) | Cortical (II, III, IV) |          |        | Total |
|--------|----------|------------------------|------------------------|----------|--------|-------|
| DP     | SWI      | 57 (71%)               | 12 (15%)               | 10 (12%) | 2 (2%) | 81    |
|        | WHAT     | 82 (81%)               | 11 (10%)               | 6 (6%)   | 3 (3%) | 102   |
| CRZ    | WHAT     | 127 (70%)              | 54 (30%)               |          |        | 181   |
| SS     | WHAT     | 160 (74%)              | 57 (26%)               |          |        | 217   |