

## Serial Susceptibility Weighted (Phase) Imaging in Multiple Sclerosis

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**Introduction:** Multiple sclerosis (MS) is a recurrent and chronic inflammatory disease of the central nervous system characterised by inflammation, demyelination, gliosis and axonal loss. Lesions originate around venous vessels [1,2] and evidence is increasing that iron plays an important role in the pathogenesis of MS [3]. Susceptibility weighted imaging (SWI) [4] displays small venous vessels and its phase images are sensitive to altered iron content [5]. The objective of this work was to apply SWI in a longitudinal study of MS.

**Subjects and Method:** Data were acquired as part of an ongoing serial study on demyelination and remyelination in MS, using T2 relaxation for myelin water imaging [6], diffusion tensor imaging (DTI), spectroscopy, SWI and standard MRI methods (FLAIR, Gd-DTPA enhanced T1-weighted imaging). 20 subjects with relapsing-remitting MS (15 female, 5 male; median EDSS = 2.5 (range 1.0-6.0); mean age = 40yrs (range 28-57yrs); mean disease duration = 8.5yrs (range 0.5-27yrs)) were scanned at 1 month intervals over 6 months (Month 0, 1, 2, 3, 4, 5, 6) on a Philips Achieva 3.0T system. SWI data were acquired with a flow-compensated 3D gradient echo method [4] (TR/TE/alpha=40/20/19, acquisition matrix = 480 x 231 x 32, reconstruction matrix 560 x 560 x 64). Phase images [7] were unwrapped [8,9] and high pass filtered. Magnitude and phase images were inspected visually and compared with FLAIR and Gd-DTPA enhanced T1 weighted images.

**Results:** Most lesions are hyperintense on SWI magnitude. None of the six Gd-DTPA enhancing lesions investigated were visible with SWI prior to enhancement. All were visible on SWI magnitude at the time of enhancement (Fig 1.) and on all subsequent scans. Visibility in the phase increased over time after GD enhancement. The involvement of venous vessels was observed in the majority of the lesions (Fig. 2,3).

**Discussion and Conclusion:** SWI allows investigation of venous vessels and potential iron deposits at the same time. That enhancing lesions become phase-hypointense up to 3 months after lesion formation (and the fact that contrast agent enhancement is due to a disruption of the blood brain barrier) suggests that blood products may accumulate and cause the lesion visibility in SWI-phase. The contrast mechanisms, e.g. why some lesions are hypointense in the magnitude of SWI and others are hyperintense, are far from being understood. We expect to gain further insight from the complete analysis, including DTI, T2 and MRS, of the whole cohort.

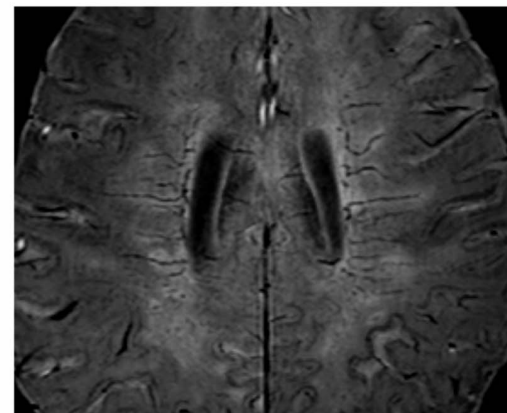
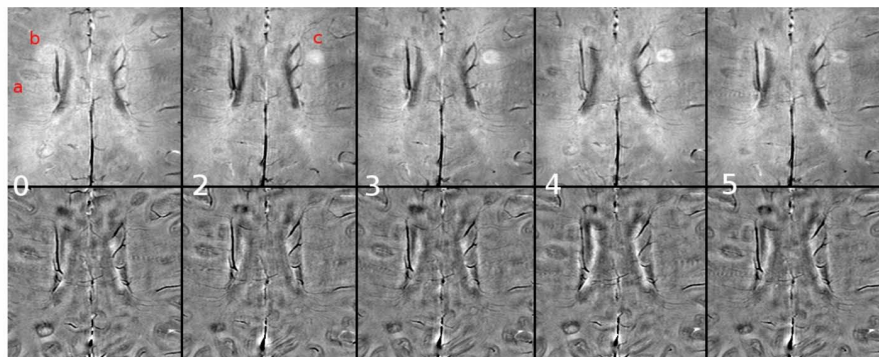


Fig 2.: SWI venograms reveal the close spatial relationship between veins and MS lesions.

Fig 1.: Magnitude (top row) and phase (bottom row) of SWI over 5 months. Lesion **a** is hypointense on the magnitude and traversed by a vein. During the 5 months lesion **b** became less hyperintense on the magnitude. Lesion **c** is contrast enhancing and appeared between month 0 and month 2. Initially, it was only visible on the magnitude. It developed a magnitude hypointense centre which became increasingly visible in the phase.

**References:** [1] Zamboni PJ Royal Soc Med 2006;99:589-593; [2] Lassmann et al. Brain Pathol. 2007 Jul;17(3):325. [3] LeVine et al. Ann. N.Y. Acad Sci 2004;1012:252-266 [4] Reichenbach JR et al. NMR Biomed. 2001;14(7-8):453-67. [5] Haacke EM et al. J Magn Reson Imaging. 2007 Aug;26(2):256-64. [6] Whittall KP et al. Magn Reson Med. 1997 Jan;37(1):34-43. [7] Rauscher et al. AJNR Am J Neuroradiol. 2005 Apr;26(4):736-42. [8] Witoszynskij S et al. Medical Image Analysis, 2008, in press [9] Rauscher et al: J Magn Reson Imaging. 2003 Aug;18(2):175-80.

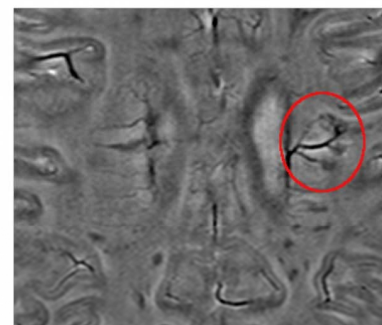


Fig 3.: SWI phase image. Venous vessels connect to four lesions, producing an atypical vascular pattern compared to the normal contralateral side.