Susceptibility Weighted Imaging Reveals Unique Information in Multiple-Sclerosis Lesions Using High-Field MRI.

E. Haacke[1], M. I. Makki[2], M. Selvan[1], Z. Latif[1], J. Garbern[3], J. Hu[1], M. Law[4], and Y. Ge[4]

[1]Radiology, Wayne State University, Detroit, Mi, United States, [2]Radiology and Neurology, Wayne State University, Detroit, Mi, United States, [3]Neurology, Wayne State University, Detroit, Mi, United States, [4]Radiology, New York University, New York, NY, United States

Introduction: Multiple sclerosis (MS) is a chronic disease of the central nervous system (CNS) manifest by a recurrent or chronic angiocentric inflammatory process resulting in myelin loss coupled with degeneration of axons. T2 shortening, probably a result of nonheme iron deposition, has been reported in MS (1). The purpose of this study is to investigate the potential of susceptibility weighted imaging (SWI) (2), which exploits the magnetic susceptibility difference between tissues, in revealing abnormal iron deposition in MS lesions that is otherwise not easily seen on conventional MRI.

Materials and Methods: Nine established MS patients underwent conventional imaging and SWI at WSU. (Patient 3 was scanned twice, the second scan being referred to below as 3b). SWI is a 3D fully flow compensated balanced GRE sequence. All scans were acquired on a 4T Bruker MEDSPEC system. Imaging parameters for this sequence were: TR/TE=24/15 ms, flip angle = 12°, matrix = 256x256, FOV=256x256 mm^2, 64 slices and slice thickness = 2mm. In a parallel study, four established MS patients underwent SWI on a 3T Siemens system at NYU. Imaging parameters for this sequence were: TR/TE=50/25 ms, flip angle = 20°, matrix = 512x512, FOV=220x220 mm^2, 32 slices and slice thickness = 2mm. Hyperintense lesions were localized in both FLAIR and T2W images first and then identified on SWI using dedicated homemade software (SPIN). Both magnitude and SWI filtered phase data were used to evaluate the presence of micro magnetic susceptibility within and surrounding the lesions. The local phase changes, supposedly due to changes in iron content, were also measured.

Results: Lesions that appear hyperintense in T2W and FLAIR are often shown as hypointense signal in phase filtered SWI (Table 1). We characterized 7 types of lesions with SWI in both studies and they are described as: (a) lesions connected by veins, (b) lesions site on venous structures, (c) lesions surrounded by a large rim with hypointense signal, (d) lesions with a hypointense center, (e) lesions seen in SWI not clearly identified with FLAIR, (f) lesions seen with FLAIR but not identified in SWI and (g) abnormal iron content in the GM seen in SWI not in conventional T2 and FLAIR.

Category	Number of lesions in each category													
Field	4T data										3T data			
Patient	1	2	3	3b	4	5	6	7	8	9	1	2	3	4
Category (a)	2	-	12	13	1	4	2	6	1	2	1	5	-	1
Category (b)	1	-	2	2	-	2	-	-	-	-	2	-	-	-
Category (c)	1	-	4	4	-	3	-	-	1	-	2	-	-	-
Category (d)	1	1	7	7	1	4	3	18	-	2	5	12	-	1
Category (e)	-	1	4	4	-	3	-	10	-	1	-	-	-	-
Category (f)	2	-	1	1	7	-	-	-	-	-	-	-	-	10
Category (g)	3	4	6	7	3	5	4	4	2	5	3	4	2	2

Discussion and Conclusion: The SWI data directly revealed and confirmed the close relationship between MS lesions and venous structures, indicating the role of inflammatory vascular pathology in MS. In addition, SWI has the potential to show additional characteristics of iron deposition in these lesions which should provide new insight into the pathophysiology of tissue damage of the disease. This may help to distinguish between different types of tissue injury and lesion activity in this highly heterogeneous disease.

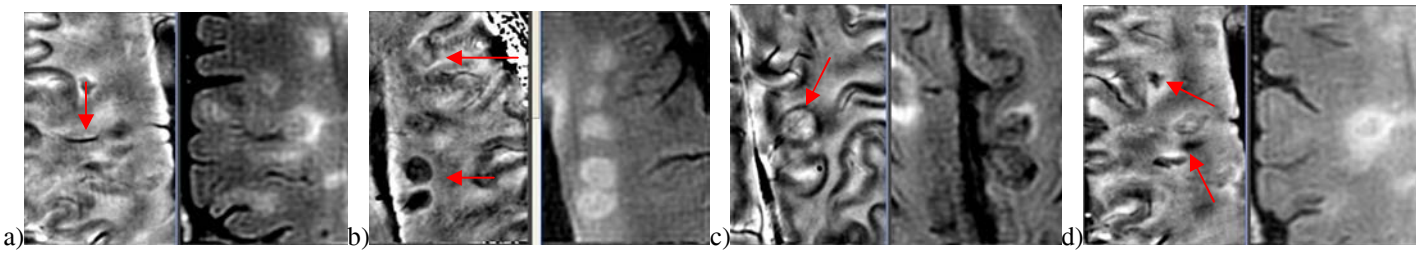

Figure 1: These images compare SWI (left) and FLAIR (right) for 4 patients. (a) SWI clearly shows vessels connecting the lesion to the pre-central gyrus and ventricle; (b) a diffuse signal abnormality (indicating increased iron deposition) in SWI compared to FLAIR; (c) SWI clearly enhances the rim of the lesion that appears smaller in FLAIR;. d), lesions seen in SWI (see arrows) but not seen with FLAIR.

References: 1) Bakshi et al., Neuroreport, 17(2000). 2) Reichenbach et al., Radiology 204(1997). 3) Law et al., Radiology 231 (2004).