

Diffusional Kurtosis Imaging of White Matter and Gray Matter Lesions in Multiple Sclerosis: Combined Use with Double Inversion Recovery

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Target audience: Radiologists, Neurologists, researchers of diffusion MRI, residents/fellows & techs.

Introduction and Purpose: Diffusion magnetic resonance (MR) metrics can quantify pathological changes in multiple sclerosis (MS) plaques, normal-appearing white matter (NAWM), and normal-appearing gray matter (NAGM).^{1,2} MS has been classically regarded as a white matter (WM) disease. However, recent histopathological studies have convincingly shown that grey matter (GM) regions are also heavily affected. Diffusional kurtosis imaging (DKI) is an extension of diffusion tensor imaging (DTI) that enables simultaneous quantification of Gaussian and non-Gaussian diffusion in the brain.³ The purpose of this study is to evaluate diffusional changes of the WM and GM including plaques, periplaque WM (PWM), periplaque GM (PGM), NAWM, and NAGM in MS by using a new method—DKI with double inversion recovery (DIR) to segment GM.

Methods: The participants were 7 MS patients with GM plaques. Diffusion-weighted images were obtained on a 3T MR imager (Achieva; Philips Healthcare) by using a spin-echo echo-planar imaging sequence with 3 diffusion weightings ($b = 0, 1000, \text{ and } 2000 \text{ s/mm}^2$) along 20 diffusion-encoding directions. Parametric maps of the standard DTI metrics of mean diffusivity and fractional anisotropy (FA) as well as the additional DKI metric of mean kurtosis (MK) were obtained. For analyses of GM, a DIR image was registered with a diffusion map by using the SPM8 software package. Thirteen plaques, these peripheral lesions (PWM, PGM), NAWM, and NAGM were analyzed. Intracortical lesions are readily detected on

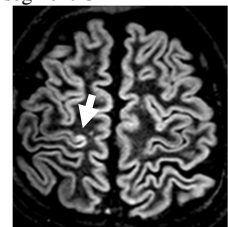
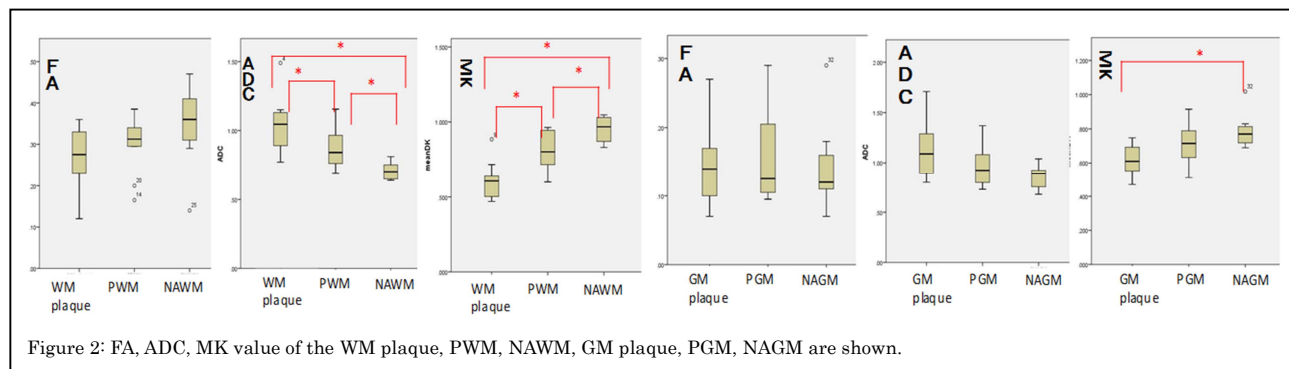


Figure1: A cortical lesion in MS patients visualized in DIR.

DIR images, as shown in the Figure 1.

Results: Significant differences were seen in ADC and DK among NAWM, PWM, and WM plaques ($*P < 0.05$, Steel–Dwass multiple comparison test). Only MK differed significantly between NAGM and GM plaques ($*P < 0.05$). These results are shown in the Figure 2.



Discussion: MK may be a more sensitive biomarker of tissue damage in MS patients compared with FA. FA and MK decrease, whereas ADC increases, in the order of NAWM, PWM, and WM plaques. There is a gradient of WM abnormality extending outward from the plaque. Typical pathological hallmarks of WM lesions such as lymphocyte infiltration and complement deposition are not usually found in cortical lesions. Meningeal inflammation is linked to cortical pathology in MS.^{2,4} Our proposed method can detect differences around WM or GM lesions. In contrast to WM plaques, there may be little change in the GM substance around GM plaques. Therefore, DKI with DIR is useful for segmenting GM lesions.

Conclusion: DKI with DIR detected abnormalities in WM and GM with high sensitivity and can provide additional information on changes of WM and GM in MS.

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