DIFFUSION WEIGHTED IMAGING IN THE ASSESSMENT OF RENAL DYSFUNCTION

L. C. Chow[1], R. Chang[1], R. Bammer[1]

[1]Radiology, Stanford University School of Medicine, Stanford, CA, United States

Synopsis: The ability of diffusion weighted imaging to reflect and lateralize renal dysfunction was assessed by comparing renal ADC with serum creatinine values.

Introduction: While diffusion-weighted imaging (DWI) has been predominantly applied in the CNS [1], recent studies have begun to explore the feasibility of DWI MRI in the abdomen and its use in detecting and characterizing abdominal abnormalities[2-8]. Until recently, attempts at abdominal DWI have been thwarted by the presence of physiologic motion including respiration, peristalsis and blood flow, which are orders of magnitude greater in amplitude than diffusional motion and can easily overwhelm the effects of diffusion. Newer, more powerful gradients yielding reductions in diffusion-encoding and EPI readout times, have allowed breath-held full-organ coverage DWI in the abdomen with improved image quality. In this study, the relationship between ADC values and renal failure was evaluated by comparing renal ADC values with serum creatinine levels in patients with normal and abnormal renal function.

Methods: Breath-held abdominal DWI was performed in 15 patients referred for abdominal MRI. Subjects were scanned axially through the organs of interest and the kidneys. All MR scans were performed on 1.5T, commercially available MRI units (Signa CV/I and TwinSpeed, GE Medical Systems, Waukesha, WI) fitted with high performance gradients (40 mT/m, t_{rise} = 268μs) and the body coil. All procedures performed were approved by the institutional review board at Stanford University. Spin-echo-based DW-sshEPI sequences with partial Fourier EPI readout were performed with the following parameters: FOV = 30 x 30 cm to 40 x 40 cm, TR of 2600 – 3200 ms, TE of 45.3 – 55.1 ms, matrix 128 x 128, bandwidth $\pm$125 kHz. Tetrahedral diffusion encoding was utilized to allow for the determination of directionally invariant ⟨D⟩ (trace) at mimimum TE for a given b-value. The b-value was 300-s/mm^2 (2 averages) and all images were averaged in magnitude mode. Isotropically diffusion-weighted images and trace maps ⟨D⟩ were generated for all scans. Values of ⟨D⟩ obtained from ROIs drawn within the renal parenchyma on ⟨D⟩ maps were retrospectively correlated with serum creatinine levels, findings from conventional T1 and T2-weighted SE and FSE images, contrast-enhanced 3D fast spoiled-gradient echo MRI images and clinical information. DWI was performed prior to gadolinium administration in all cases.

Results: 6 out of 15 patients had abnormally elevated (> 1.6 mg/dL) serum creatinine (Cr) levels. These patients demonstrated significantly decreased ⟨D⟩ values for the kidneys, when compared with renal ⟨D⟩ values obtained in patients with normal Cr $\leq$ 1.6 mg/dL (*Figure 1*) (p=0.0005) using a two-tailed t-test with a linear correlation between ADC and Cr (r=-.741, p<0.001). Image quality was deemed excellent in all cases and in no case did susceptibility or other artifact obscure the kidneys (*Figure* 2). Two patients with borderline elevation of Cr (1.5 and 1.7) had unilateral renal disease (right renal obstruction and left renal artery stenosis, respectively) (*Figure 3)*. Renal ⟨D⟩ values were markedly asymmetric in these patients with a 23-24% reduction in ADC in the affected kidney and normal ADCs in the contralateral kidney. In patients with unilateral disease, ADCs of the affected kidneys were even lower than those of kidneys with bilateral disease. Excluding the patients with unilateral disease, the average ADCs obtained in the renal parenchyma of patients with normal Cr was 2437$\pm$111 x 10^{-6} mm^2/s (range 2281 x 10^{-6} to 2716.5 x 10^{-6}). Average ADCs obtained in the renal parenchyma of patients with elevated Cr was 2171$\pm$214 x 10^{-6} mm^2/s (range 1916 x 10^{-6} to 2279 x 10^{-6}). Differences in ADC between the right and left kidneys ranged from 3 x 10^{-6} to 266.5 x 10^{-6} with a mean of 100.9 ± 90. x 10^{-6}, excluding the two patients with unilateral disease and one patient who had only one kidney. ⟨D⟩ values for the 8 patients with Cr $\leq$ 1.6 and symmetric kidneys correlated well with normal values established in prior work[9].

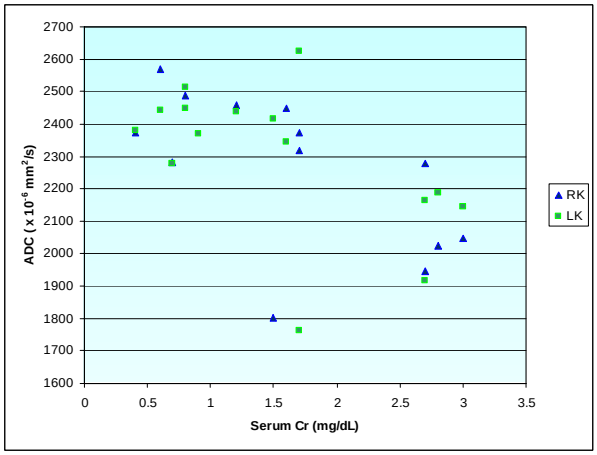


***Figure 1**. Scatter-plot demonstrating the relationship between serum creatinine levels and renal ADC. Patients with elevated serum creatinine had statistically significantly lower renal ADCs. Two patients had unilateral renal disease, which was reflected as markedly decreased ADCs on the affected side despite only borderline elevation of Cr and normal contralateral renal ADCs.*

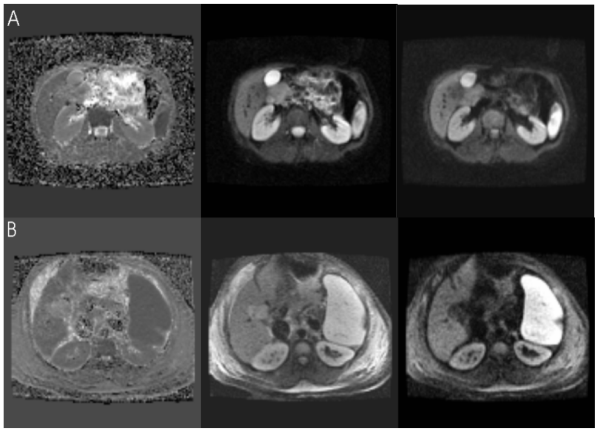
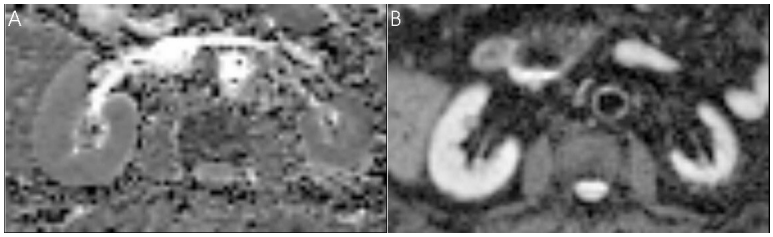


***Figure 2**. ADC map, b=0 reference images, and b=300 diffusion-weighted images in patients with normal (A) and elevated (B) serum creatinines.*

***Figure 3.** ADC map (A) and diffusion-weighted image (B) in a patient with left renal artery stenosis. Left kidney is atrophic, with visibly decreased signal-intensity on the ADC map when compared with the right. Left renal ⟨D⟩ values were markedly decreased. Right kidney ⟨D⟩ values were similar to those seen in patients with serum creatinine below 1.7 mg/dL, reflecting differences in function between the two kidneys.*



Conclusion: There is a significant decrease in renal ADC in patients with renal insufficiency and a linear correlation between renal ADC and serum creatinine in patients without unilateral disease. There was a striking difference in ADCs between normal and compromised kidneys in patients with unilateral renal disease. This technique has great potential for the evaluation of patients with renal disease, particularly those with unilateral disease such as renal artery stenosis and congenital hydronephrosis which may not be reflected in cruder measures of renal function such as serum creatinine.

References: [1] Moseley ME, *et al*. AJNR **11**: 423-9, 1990. [2] Muller MF, *et al*. Radiology **190**: 475-8, 1994. [3] Namimoto T, *et al*., Radiology: **204**: 739-44, 1997. [4] Ishikawa T, *et al*. AJR **170**: 397-2, 1998. [5] Moteki T, *et al*. JMRI **5**: 187-191, 1995. [6] Stahlberg F, *et. al*. Acta Radiol **40**: 339, 1999. [7] Ries M, *et al*. *JMRI* **14**: 42-9, 2001. [8] Murtz P, *et al*. Radiology **224**: 258-4, 2002. [9] Chow LC, *et al.* JMRI **18**: 377-382, 2003.

This work was supported in part by the National Institutes of Health (1R01EB002771 and R01-DK48051)