Evaluation of renal oxygenation in patients with renal disease using 3.0T BOLD MRI

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Introduction

The kidneys receive 25% of the cardiac output, which is the highest in the body with respect to organ weight (1). Most of the blood flow passing through the kidney is directed towards the cortex to facilitate glomerular filtration and reabsorption of the solute. Low blood flow to the renal medulla creates the osmotic gradients necessary for urinary concentration and water conservation. Moreover, the large amount of oxygen use in the ascending thick rim causes the medulla to be more hypoxic (2). A low renal medullary oxygenation level is thought to be a predisposing factor in the development of ischemic disease, such as acute renal failure (3).

Blood oxygen level-dependent (BOLD) MRI is a promising modality for monitoring renal pO2 by measuring R2*. Evaluation of normal renal medullary hypoxemia (4) and clinical application (5,6) with BOLD MRI were tried in a few studies. In this study, we measured R2* values in patients with variable medical renal disease, and tried to correlate R2* and serum creatinine level.

METHODS

From 2008 to 2010, twenty patients with renal disease were enrolled in this study. Eleven patients were referred as acute renal failure (ARF), and the rests had variable renal diseases (four patients with chronic renal failure, two patients with renal infarct, the rests were fibromuscular dyplasia, diabetes and nephrotic syndrome). MRI was performed on a 3.0 T MRI unit (Intera Achieva, Philips). Multi GE-EPI sequence with seventeen echoes was used. The scan parameters were as follows: TR, 39 ms; TE,14–39 ms with an inter-echo spacing time of 1.47 ms; slice thickness, 5 mm; flip angle, 30°; field of view (FOV), 360 mm; acquisition matrix, 256 x 256; and bandwidth, 779.5 Hz. T2* maps were generated by using IDL-based PRIDE research software (Philips Medical Systems) in a standard workstation. One cortical and one medullary ROI were drawn manually at the lower, middle and upper pole, respectively for each kidney (Fig 1). After generation of the T2* map, the R2* was calculated. We compared the means of R2* in groups among acute renal failure patients, non renal failure patients, and normal reference. And the level of creatinine at imaging was correlated with cortical and medullary R2* values respectively.

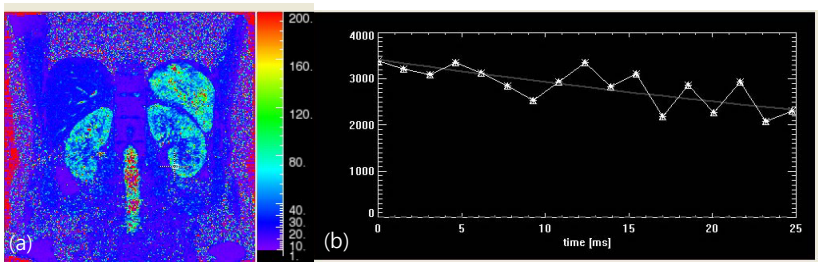

Fig 1. (a) A ROI was placed in the cortex of left kidney lower pole. (b) A T2* relaxation time curve is generated. Calculated R2* was 15.6 ms^{-1}.

RESULTS and Discussion

BOLD MRI was successful in nineteen patients. One patient was excluded from the analysis due to gross artifact interfered with R2* measurement. The mean R2* values were 13.5 ± 2.0 (Cortex) / 16.8 ± 3.9 (Medulla) in renal failure patients group, while 20.5 ± 6.0 (Cortex) / 27.3 ± 6.9 (Medulla) in non renal patients group (Fig 2.). Pearson correlation coefficient between medullary R2* values and serum creatinine level at imaging was -0.5273, showing fair negative correlation (Fig 3). Relatively small number of the patients, nonhomogeneous clinical setting of various patients and different hydration status can be limitations of our study.

In conclusion, Renal BOLD MRI can be efficiently performed with 3.0 T MRI in patients. Renal medullary hypoxia is decreased in patients with acute renal failure patients. Medullary R2* can be an indicator of decreased renal function.

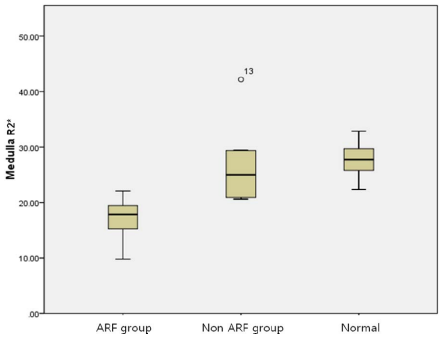

Fig 2. Mean R2* value from ARF group was significantly lower ($p<0.05$) than R2* from non ARF group.

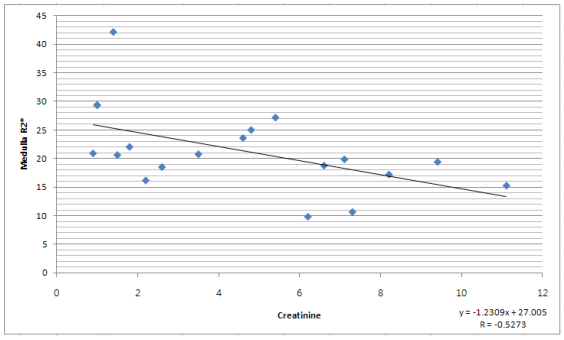

Fig 3. Medullary R2* values and serum creatinine level showed fair negative correlation (r=-0.5273).

References

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