

What Causes Diminished Corticomedullary Differentiation In Renal Insufficiency?

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Introduction

On T1-weighted MR images of the normal healthy kidney, the signal intensity of renal cortex is typically higher than medulla, resulting in easily visualized corticomedullary differentiation (CMD). Loss of CMD has been observed in renal insufficiency secondary to a variety of etiologies, such as glomerulonephritis, acute tubular necrosis, end-stage chronic renal failure, obstructive hydronephrosis, and acute allograft rejection (1-3). While average T1 values for the cortex and medulla in normal kidneys have been reported (882 ± 59 msec and 1163 ± 118 msec, respectively, at 1.5T, for example (4)), to our knowledge, the underlying changes in T1 that result in loss of CMD in renal insufficiency have not been reported. Our purpose was to investigate whether the loss of CMD associated with decreasing renal function (as measured by single kidney glomerular filtration rate (SKGFR)) is attributable to changes in T1 values of the cortex, medulla, or both.

Methods

Study subjects included 10 patients (ages 69.8 ± 16.9 yrs; 20 kidneys) referred for suspected renovascular disease. Serum creatinine exceeded 1.5 mg/dl in 4 subjects. All subjects underwent same day ^{99m}Tc-DTPA renography, where 1hr and 3 hr blood clearance and gamma camera images were used to determine SKGFR. Based on background- and attenuation-corrected renal uptake of ^{99m}Tc-DTPA in the left (U_{left}) and the right (U_{right}) kidney at 2-3 minutes, split renal function was determined as U_{left}/(U_{left}+U_{right}) and U_{right}/(U_{left}+U_{right}). SKGFR was calculated by multiplying split renal function for each kidney by global GFR (5).

T1 measurements were performed using a modified cine IR segmented k-space true fast imaging with steady-state precession (trueFISP) sequence with 6 mm thick slices (6). Images were acquired at 1.5 T (Avanto, Siemens) where the time to the first inversion was 80 msec, and subsequent inversion time (TI) intervals were 90.2 msec. Regions of interest (ROIs) were placed over the cortex and medulla in both the right and left kidney. T1 calculations were performed by fitting ROI data with the following equation:

$$M(TI) = M_0 \frac{1 + \exp(-\tau/T_1) - 2 \exp(-TI/T_1)}{1 + \exp(-\tau/T_1)}$$

where τ is the known (4 sec) time between the 10-ms adiabatic inversion pulses. A complete 180-degree inversion was assumed.

Results

Single kidney GFR values ranged from 3.49 to 89.37 ml/min based on radionuclide studies. T1 values ranged from 870 to 1329 msec in the cortex and 1085 to 1500 msec in the medulla. The average difference in T1 relaxation times between the medulla and cortex in these patients was 147.9 ± 176.0 msec, ranging from -208 to 408.5 msec. Regression analysis showed a negative relationship between cortex T1 and SKGFR (slope = -2.9, 95% CI [-5.4 to -0.4]; r = 0.5, p = 0.03), whereas there was no significant correlation between medullary T1 and SKGFR (r = 0.24, p = 0.3) (Figure 1). The difference between medullary and cortical T1s did correlate significantly with SKGFR (r = 0.58, p < 0.01) (Figure 1). These results remained unchanged after covarying for age and gender.

Conclusion

The loss of CMD has been associated with renal insufficiency secondary to a variety of etiologies. Our study shows that this loss of CMD is due primarily to an increased T1 relaxation time of the cortex. The loss of CMD seen in renal insufficiency can be due to a variety of causes depending on etiology, e.g. edema, renal atrophy, and renal scarring (1,7). It remains to be studied whether changes in cortical T1 vary across different underlying etiologies of renal dysfunction.

References

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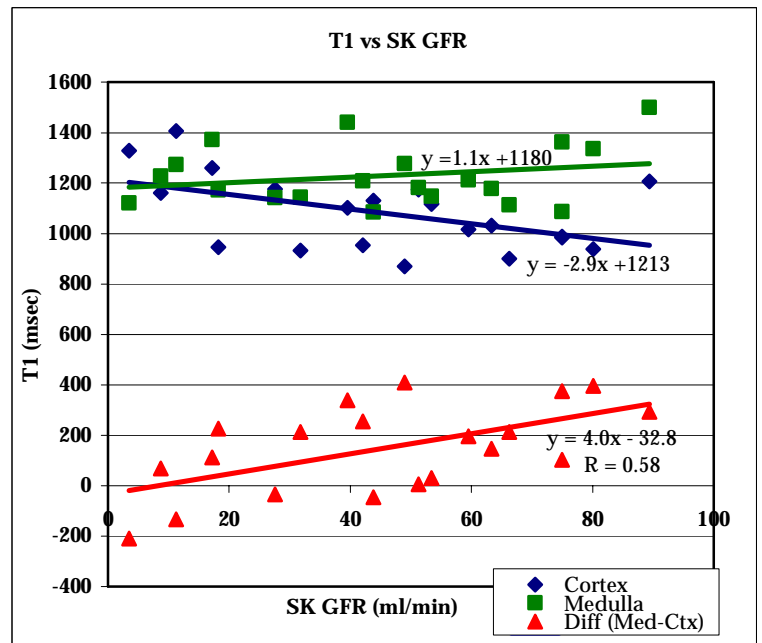


Figure 1: T1 for Cortex and Medulla vs. Single Kidney GFR