

Evaluation of Dysfunctional Renal Transplants Using Low-Dose MR Renography

L. Bokacheva¹, H. Rusinek¹, K. Prince¹, M. Kaur¹, T. Diflo², D. John², J. Bernstein³, L. Barisoni-Thomas⁴, and V. Lee¹

¹Department of Radiology, New York University School of Medicine, New York, New York, United States, ²Department of Surgery, New York University School of Medicine, New York, New York, United States, ³Department of Medicine, New York University School of Medicine, New York, New York, United States, ⁴Department of Pathology, New York University School of Medicine, New York, New York, United States

Introduction

Dynamic contrast-enhanced (DCE) imaging of the kidneys (MR renography, MRR) enables assessment of the renal function and morphology. Standard-dose gadolinium-enhanced MRR has shown a good promise as a tool for evaluation of renal transplants [1-3]. Given concerns about nephrogenic systemic fibrosis, we have examined the use of low-dose MRR with 4 ml Gd-DTPA [4] to study the differences among various causes of graft dysfunction.

Methods

We examined 6 normally functioning renal transplants and 11 transplants during dysfunctional episodes. Diagnoses were established by renal biopsy or by consensus assessment of all clinical data by a team of 2 experienced transplant surgeons, nephrologist, and renal pathologist and included acute graft rejection (n=6) and delayed graft function/acute tubular necrosis (ATN) (n=5). Imaging was performed at 1.5 T (Avanto or Symphony, Siemens) with 3D FLASH oblique coronal sequence (typical parameters TR/TE/flip angle=2.84/1.05/12°, 1.7x1.7x2.5 mm³ voxel, 3 s acquisition time) acquired repeatedly for 10 min after a bolus of 4 ml Gd-DTPA and 20 ml saline flush, injected at 2 ml/s. Renal images were co-registered and segmented into cortex and medulla with the semiautomatic algorithm [5]. Signal intensity values were extracted and converted into gadolinium concentration using GRE formula and pre-contrast T₁ values measured by low flip angle TrueFISP [6]. The cortical and medullary concentration versus time curves were evaluated, and maximum cortical concentration during the vascular phase, and cortical and medullary concentrations at t=120 s after the start of the acquisition were compared.

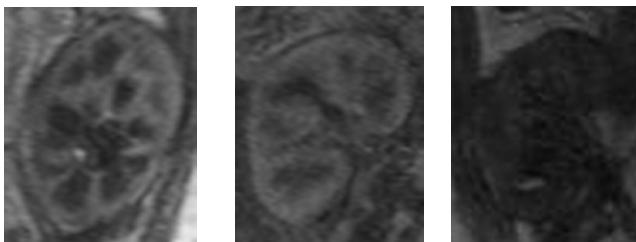


Fig. 1: MRR images of a normally functioning kidney transplant (left), kidney with ATN (middle), and kidney suffering from rejection (right), at t=12 s after the start of acquisition.

curves, although their vascular and tubular features are broadened. In more severe cases, such as shown in Fig. 2c, no vascular peaks are observed, and the difference between cortex and medulla is small. The maximum cortical concentration in ATN cases (mean 0.15 mM) is significantly lower than in normal kidneys (mean 0.26 mM, t=3.05, P=0.014) or in rejected kidneys (mean 0.30 mM, t=4.44, P=0.002). In ATN cases, cortical (mean 0.076 mM) and medullary concentrations (0.087 mM) at t=120 s are also lower than in normal kidneys (cortical 0.20 and medullary 0.25 mM, respectively) (P=0.0016 and P=5·10⁻⁴). The differences between concentrations in normal and rejected kidneys are not significant.

Discussion

Using low-dose MRR, we observed that characteristic cortical and medullary enhancement curves in normal transplants show contrast passing through the renal vasculature (vascular peaks, t=20 s) and into the renal tubules after glomerular filtration (tubular peaks, t>60 s), where the most prominent feature associated with the tubular transport is the medullary peak observed at about t=120 s. In ATN cases, the medullary peak is not present, while the vascular peaks may be observed in some cases. This suggests that the MRR curves reflect the tubular injury. Mild rejection cases are difficult to separate from normal transplants, yet severe rejection is manifested as dramatically diminished enhancement and poor differentiation between cortex and medulla.

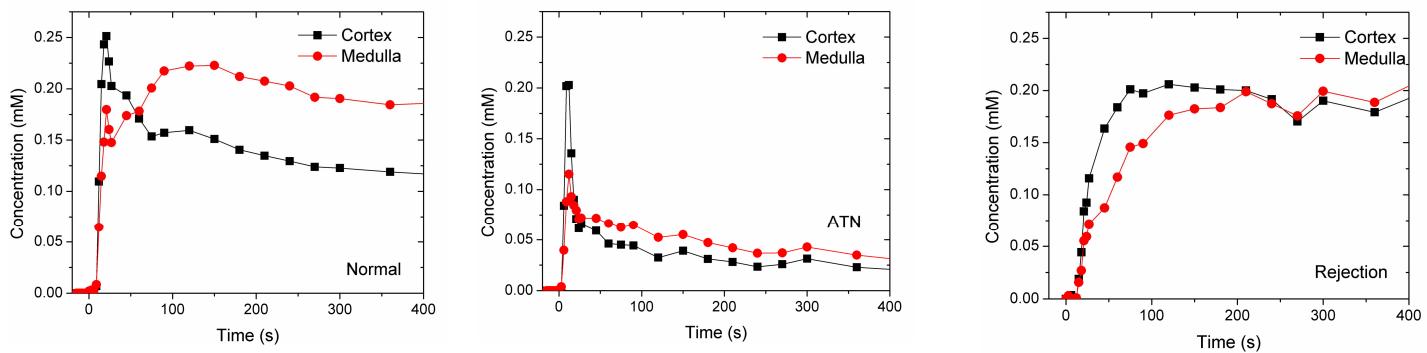


Fig. 2: Cortical and medullary concentrations for a normally functioning graft (left), graft with ATN (middle), and rejected graft (right).

References

[1] Sharma RK et al. Transplantation 1995;59:1405-1409. [2] Szolar DH et al. MRI 1997;15:727-735. [3] Neimatallah M et al. JMRI 1999;10:357-368. [4] Lee VSL et al. Am J Physiol Renal Physiol 2007;292:1548-1559. [5] Rusinek H et al. MRM 2007;57:1159-1167. [6] Bokacheva et al. MRM 2006;55:1186-1190.