Quantitative Longitudinal Relaxation Time (T1) As Part of Multiparametric MRI Mapping Reveals Distinct Patterns of Renal Injury in AKI and CKD

**PURPOSE:**

Acute Kidney Injury (AKI) and Chronic Kidney Disease (CKD) both result in a reduction in GFR but by different mechanisms. Nevertheless, there is growing evidence of a relationship between AKI and CKD such that incomplete recovery of AKI may result in CKD. The pathophysiology of progression from AKI to CKD in humans is not well described. Here, as part of a multiparametric MRI study, longitudinal relaxation time (T1) mapping was performed to assess differences in renal structure and function between AKI and CKD.

**METHODS:**

57 participants; 10 AKI patients, 26 CKD patients and 21 healthy volunteers (HV) were included in this study. AKI patients (inpatient AKI stage 2/3, no pre-existing CKD) underwent MRI scans at the time of AKI, 90 days later and a year later. Patients with CKD stage 3/4 underwent MRI within 3 months of a routine biopsy; their mean eGFR was 39±13 ml/min/1.73 m2 and urine protein:creatinine ratio 120±188mg/mmol. HVs were age matched, had normal renal function and no proteinuria. MRI scans were performed on a 3T Philips Ingenia scanner. MR measures of structure and function were taken, including T1 mapping as a marker of fibrosis and/or inflammation.

**RESULTS:**

Table 1 demonstrates T1 values within the cortex and medulla for HVs, AKI patients at time of injury and at 3 months post-AKI, and CKD patients. AKI patients at the time of injury had the highest T1  values in both the renal cortex and medulla; at 3 months post-AKI both cortical and medullary T1 values had declined but remained elevated comapred to HV levels. Interestingly, at 3 months post-AKI cortical T1 declined to a value similar to that of CKD patients, whilst medullary T1 remained significantly higher than in CKD patients T1

Table 1.

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | **HVs** | **AKI Injury** | **AKI 3 Months** | **CKD** |
| **T1 Cortex (ms)** | 1416± 79 | 1738±23 | 1546±21 | 1554±96 |
| **T1 Medulla (ms)** | 1690±86 | 2043±78 | 1919±83 | 1738±74 |

There were differences between AKI and CKD patients in the correlation of T1 values and biochemistry and histology variables. In AKI patients, higher T1 values in the medulla correlated with larger changes in serum creatinine between peak inpatient value to creatinine at the first AKI scan (r = 0.71, p = 0.05), and between first scan and second scan (r=0.844, p=0.02). This pattern of high medullary T1 values was not seen in CKD, instead correlations with clinical variables were observed with T1 measurements in the cortex: cortical T1 correlated negatively with eGFR (r=-0.6, p=0.002) and positively with interstitial fibrosis score (r=-0.52, p=0.02).

**DISCUSSION:** This comparative study demonstrates that longitudinal relaxation time (T1) mapping is able to identify important pathophysiological processes in AKI and CKD, and can demonstrate clear differences in the pattern the of injury between the two. T1 changes were more marked in the medulla of patients with AKI, with increased T1 values suggesting oedema or inflammation at time of AKI. In contrast, cortical changes were more closely correlated with important clinical parameters in CKD. Results also suggest that MRI has the potential to inform the transition from AKI to CKD. Incomplete recovery in MRI measures was seen at 90days after AKI, at which time the pattern of abnormalities more closely resembled those of CKD patients. Further longitudinal studies are underway to investigate MRI changes in AKI and CKD and their clinical utility.