Multiparametric Renal MRI in Chronic Kidney Disease: changes in clinical and MRI measures over two years

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Background and Aims: Chronic kidney disease (CKD) progression is currently monitored using estimated GFR (eGFR) and albuminuria but these are relatively crude measures with multiple limitations. Multiparametric renal magnetic resonance imaging (MRI) combines several MR measures into a single scan session, offering the potential to assess severity, progression and response to therapy in CKD in a novel, non-invasive way. Previous studies have focussed on cross-sectional comparisons of MRI and clinical measures. In this study we sought to investigate the ability of MRI variables to predict and monitor progression of CKD over two years.

Methods: Persons with CKD stage 3-4 (eGFR 15-59ml/min/1.732) who had undergone renal biopsy were recruited. Participants underwent multiparametric renal MRI scans on a 3T Philips Ingenia scanner at baseline, one and two years. In addition demographic data, medical history, eGFR and urine protein:creatinine ratio (uPCR) was collected. Multiparametric renal MRI comprised longitudinal relaxation time (T1 using SE-EPI and bFFE readouts), Diffusion Weighted Imaging, renal blood flow (Phase Contrast MRI), renal cortex perfusion (Arterial Spin Labelling), and Blood Oxygen Level Dependent (BOLD) relaxation rate (R2*). CKD progression was defined as participants having a slope in eGFR of -5ml/min/yr or greater over 2 years. Interstitial fibrosis was quantified on renal biopsy samples.

Results: 22 participants underwent MRI scanning at baseline (7 participants were classified as ‘progressors’ and 15 as ‘stable’), while 13 completed all three scans (4 of whom were ‘progressors’). At baseline, cortex T1 was significantly higher for ‘progressors’ compared to ‘stable’ participants (p=0.02), and renal cortex perfusion was significantly lower (p=0.03). There was no significant difference in total kidney volume (TKV), ADC, renal cortex or medulla R2*, or renal biopsy measures of interstitial fibrosis between ‘progressor’ and ‘stable’ CKD participants.

At Year 1 and Year 2 compared to baseline, a decrease in total kidney volume (TKV) was found, with a significantly greater decrease in TKV in the ‘progressor’ group (p=0.04). Over time, T1 increased in the ‘progressor’ group versus baseline, particularly in the cortex which showed a significant difference at year 1 (p=0.034) and year 2 (p=0.053). There was an effect for a reduction in ADC in ‘progressors’ versus stable participants over time. There was no significant change over time or between groups in renal cortex perfusion, renal cortex or medulla T2*, or renal biopsy measures of interstitial fibrosis.

Conclusion: Our results show that at baseline lower renal cortex perfusion and higher renal cortex T1 were associated with progression of CKD over 2 years suggesting that these MRI parameters may be a predictors of progression. On the other hand, cortex T1, TKV and ADC changed more in ‘progressors’ than in ‘stable’ participants over time (TKV and ADC decreased, T1 increased), suggesting that they may be useful MRI measures to monitor progression. Further studies are required to confirm these findings in a larger cohort of patients before renal MRI can be recommended for clinical use.