Multiparametric Magnetic Resonance Imaging Assessment of Chronic Kidney Disease

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Introduction

Progression of Chronic Kidney Disease (CKD) occurs as a result of a common final pathway of inflammation and fibrosis that may be independent of the underlying aetiology.

In current clinical practice, assessment of renal parenchymal damage is confined to renal biopsy, which is limited by sampling error and is associated with patient risk. Recent advances in Magnetic Resonance Imaging (MRI) allow assessment of structural and functional changes relevant to kidney disease. We performed a multiparametric MRI study to assess its utility and reproducibility in patients with CKD.

Methods

39 people were studied: 26 patients with CKD Stage 3-4 who had undergone renal biopsy as part of routine clinical care, and 13 healthy volunteers (HVs) as a comparator group. Patients had two multiparametric renal MRI scans performed 7-14 days apart and HVs underwent a single multiparametric MR scan. Biochemical and clinical parameters were collected at the first scan.

MRI scans were performed on a 3T Philips Ingenia scanner. Functional assessment included kidney volume, longitudinal relaxation time (T_1) mapping and diffusion weighted imaging (DWI) to compute apparent diffusion coefficient (ADC) and cortex D as markers of fibrosis and/or inflammation. Arterial Spin Labelling (ASL) was used to measure renal perfusion, phase contrast angiography (PCA) to measure blood flow in the renal artery and Blood Oxygenation Level Dependent (BOLD) Imaging was used as a measure of renal oxygenation. Coefficient of variance (CoV) was calculated for each MRI measure between the two scan days. Biochemical data, and biopsy glomerular sclerosis(GS) and interstitial fibrosis(IF) were correlated with MR parameters

Figure 1 A, Illustrates the Study Protocol

Figure 1B, Illustrates the Multiparameteric MR protocol (One hour)

Results

The 26 CKD patients had a mean age of 57±15yrs, 20 were male, and mean estimated glomerular filtration rate (eGFR) was 39±13 ml/min/1.73 m^2. Mean urine Protein Creatinine Ratio (PCR) was 120±188mg/mmol. Seven patients had ischaemic nephropathy, five had tubulointerstial disease and 14 had glomerulonephritis, biopsies were performed 53 (13 - 98) days before the first scan. HVs had a mean age 56±19yrs, nine were male and all subjects had an eGFR>60mls/minute/1.73m^3 and urine PCR <15mg/mmol.

CKD patients had higher T_1 (measured using spin-echo echo planar imaging) and lower ADC and D (restricted movement of water molecules and subsequently another marker of fibrosis/inflammation) values than HVs (figure 2B). CKD cortical and medullary T_1 values were 1580±92ms and 1739±78ms respectively compared to HVs of 1396±64 and 1700±107 (p<0.0001) respectively (figure 2A).

Renal perfusion was lower in patients with CKD (figure 2C). There were no differences in renal volume and T_2* (BOLD) between the CKD and the HV group.

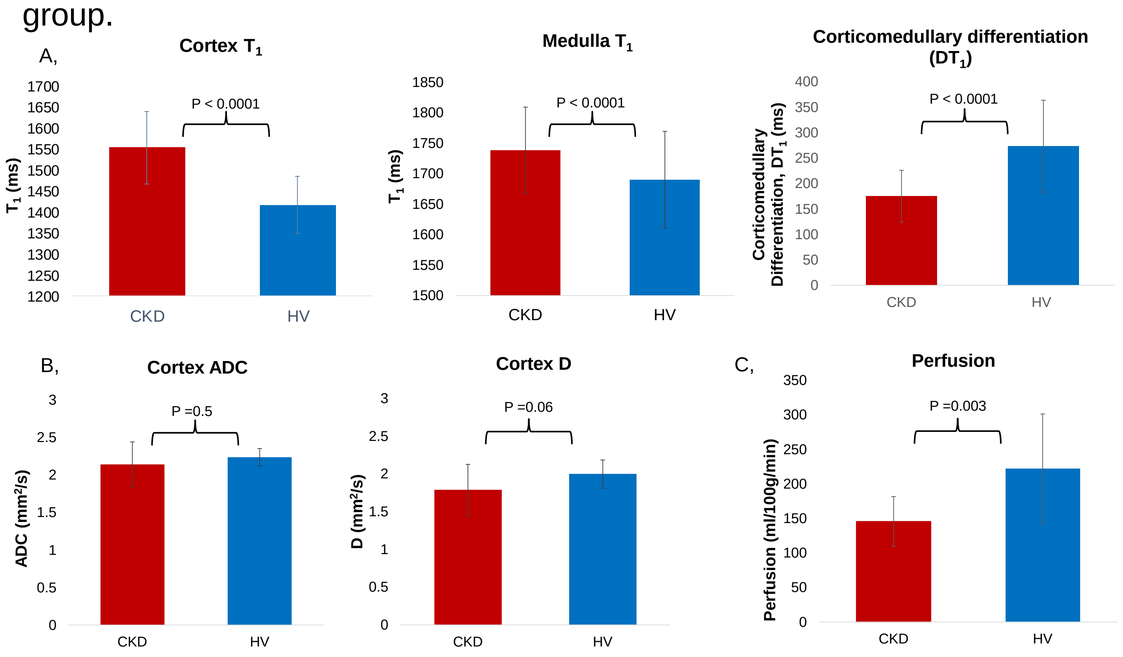
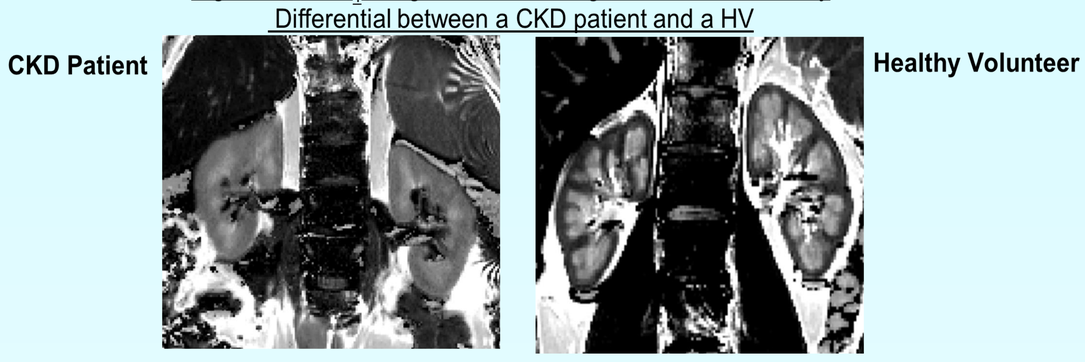


Figure 2 A, Illustrates the difference in T_1 values between the CKD and HV group
Figure 2B, Illustrates the difference in diffusion weighted imaging (ADC and D values) between the CKD and HV group
Figure 2C, Illustrates the difference in perfusion between CKD and HV group

Figure 3, MR T_1 Image Demonstrating the Corticomedullary Differential between a CKD patient and a HV

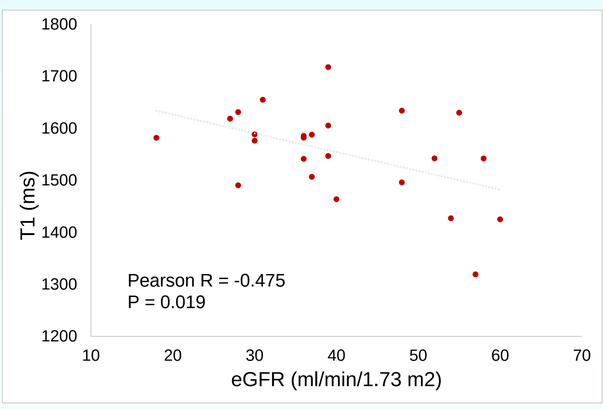


The mean T_1 corticomedullary differentiation was reduced in CKD patients compared to healthy volunteers; 192±47ms versus 334±63ms (Figure 2A, example images shown in Figure 3).

Table 1, Illustrates the reproducibility of the individual MR parameters

MRI Measure		Coefficient of Variance (%)	
		CKD	HV
T_1	Cortex	2.4	2.2
	Medulla	1.7	2.1
	Corticomedullary Differentiation	20.0	9.2
Cortex ADC		6.6	2.9
Cortex D		9.0	10.3
Total kidney Volume		2.4	2.1
T_2* (BOLD)		2.7	6.4
Perfusion		26.0	9.1

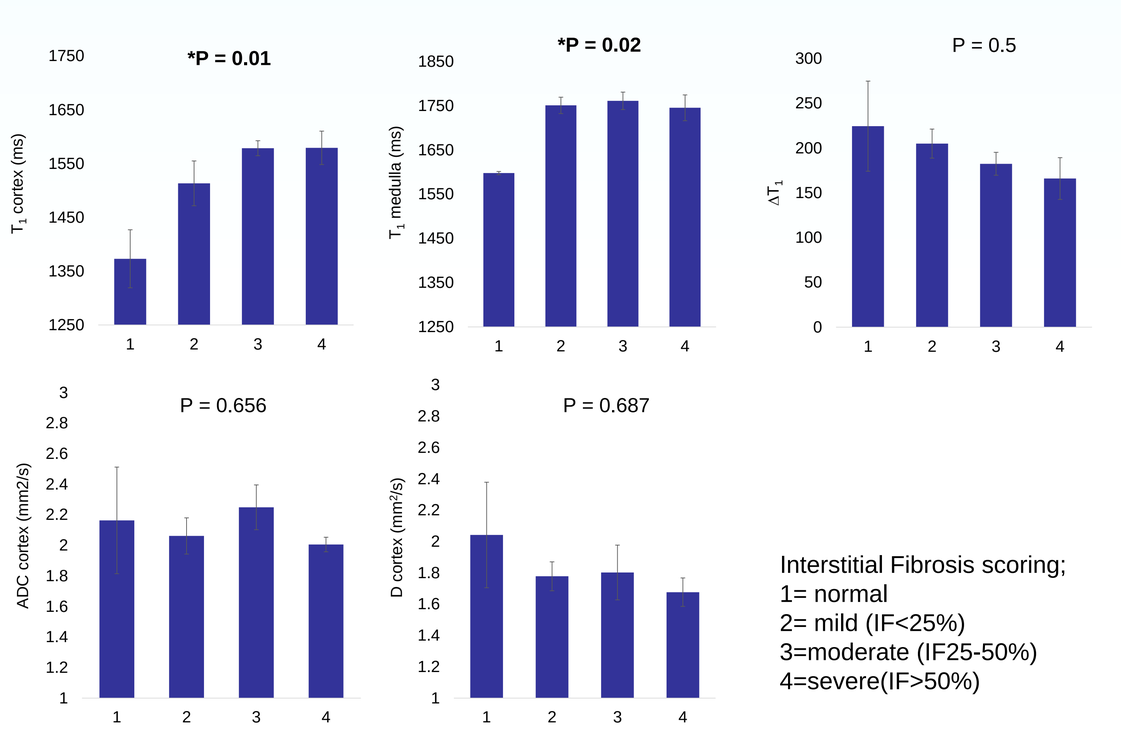
Figure 4, Correlation of T_1 with eGFR in CKD



Cortical and medullary T_1 measurements, T_2*, kidney volume and ADC all had coefficients of variance <10% in CKD patients, (table 1). There was a negative correlation between cortical T_1 and eGFR; higher T_1 values were associated with lower eGFR values.

The severity of IF biopsy score correlated with higher T_1 medullary and cortical values and lower corticomedullary differentiation. There was no correlation between ADC or D with IF. There was also no correlation between T_1 values and GS severity(figure 5).

Figure 5, Interstitial Fibrosis Scores Plotted Against Different MR Parameters



Interstitial Fibrosis scoring;
1= normal
2= mild (IF<25%)
3=moderate (IF25-50%)
4=severe(IF>50%)

Discussion

This study demonstrates that multi-parameteric MRI is highly reproducible, and shows clear differences between HVs and CKD patients. Significant differences were seen between the HVs and CKD patients in perfusion, cortical, medullary & corticomedulary T_1 and diffusion weighted imaging (D) values.

Importantly, results indicate that MRI measures are able to detect specific renal pathophysiological changes, in particular increased T_1 times correlating with interstitial fibrosis scores. The reduction in perfusion confirms previous similar findings in patients with CKD, but interestingly there was no difference in T_2*, a potential marker of renal hypoxia.

Further work is planned to assess the longitudinal change in renal function and MRI measures, and more detailed categorising of biopsy characteristics to correlate against MR parameters.

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CKD - Pathophysiology & progression II
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DOI: 10.3252/pso.eu.54ERA.2017
ePosters supported by F. Hoffmann- La Roche Ltd.