Non contrast renal magnetic resonance imaging to assess perfusion and corticomedullary differentiation in health and chronic kidney disease

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Abstract

Aims

Arterial spin labeling magnetic resonance imaging (ASL MRI) measures perfusion without administration of contrast agent. Whilst ASL has been validated in animals and healthy volunteers, application to chronic kidney disease (CKD) has been limited. We investigated the utility of ASL MRI in patients with CKD.

Methods

We studied renal perfusion in 24 healthy volunteers (HV) and 17 patients with CKD (age 22–77 years, 40% male) using ASL MRI at 3.0T. Kidney function was determined using estimated glomerular filtration rate (eGFR). T1 relaxation time was measured using MOLLI and FAIR True FISP was performed to measure cortical and whole kidney perfusion.

Results

T1 was higher in CKD within cortex and whole kidney, and there was association between T1 time and eGFR. No association was seen between kidney size and volume and either T1, or ASL perfusion. Perfusion was lower in CKD in cortex (136±37 vs 279±69ml/min/100g; $p<0.001$) and whole kidney (146±24 vs. 221±38ml/min/100g; $p < 0.001$). There was significant, negative, association between T1 longitudinal relaxation time and ASL perfusion in both the cortex ($r = -0.75$, $p < 0.001$) and whole kidney ($r = -0.50$, $p < 0.001$). There was correlation between eGFR and both cortical (0.73, $p <0.01$) and whole kidney ($r = 0.69$, $p<0.01$) perfusion.

Conclusions

Significant differences in renal structure and function were demonstrated using ASL MRI. T1 may be representative of structural changes associated with CKD, however further

investigation is required into the pathological correlates of reduced ASL perfusion and increased T1 time in CKD.

Key words

Magnetic resonance imaging

Arterial spin labeling

Renal perfusion

Chronic kidney disease

Introduction

Renal perfusion is an important physiological parameter in health and disease. In normal physiology, renal blood flow is an important determinant of oxygen supply and glomerular filtration rate [1] whilst in chronic kidney disease (CKD), renal microvascular dysfunction is one of a number of common pathological mechanisms involved in the progression of disease, irrespective of the initiating insult.

Despite this crucial role of perfusion in renal physiology and disease, *in vivo* measurement remains a challenge in both clinical and research settings, as established methods are associated with a number of inherent drawbacks. Measurement of the clearance of para-aminohippuric acid (PAH) is time consuming and invasive [2], whilst computed tomography (CT) and nuclear medicine techniques carry a radiation burden, with the former requiring administration of nephrotoxic iodinated contrast. Dynamic contrast enhanced (DCE) magnetic resonance (MR) techniques can be used to measure renal perfusion but the administration of gadolinium-based agents is now relatively contraindicated in patients with renal impairment, due to an association with nephrogenic systemic fibrosis [3].

Arterial spin labeling (ASL) magnetic resonance imaging (MRI) is an imaging technique allowing non-invasive measurement of renal perfusion using magnetically labelled blood as a contrast agent. Protons in blood are labelled by application of a saturation, or inversion, radiofrequency pulse, which then alter tissue magnetization upon exchange with blood within capillary beds. An unlabelled image is also acquired, and the ASL signal is determined by subtraction of the two. ASL MRI has an inherently low signal-to-noise ratio, due to the low contribution of inflowing blood to total tissue magnetisation. Nevertheless, ASL MRI has been validated in animals against a microsphere technique [4], and in an explanted kidney model [5], with close correlation observed between methods. In humans, good reproducibility

has been confirmed in healthy volunteer studies [6]. For example, in a recent study a coefficient of variance of 9.2% and 7.1% for cortical perfusion and whole kidney perfusion was demonstrated [7]. A small number of studies have shown reduced perfusion in patients with CKD compared to controls [8,9], and in poorly functioning kidney transplants compared to transplants with better function [10-12].

Nevertheless, ASL MRI has not yet entered widespread clinical use, hampered by lack of standardization in sequence acquisition protocols, and post processing methods. The utility of ASL MRI as a marker for disease severity and progression in CKD, and as a measure of response to therapy, is yet to be determined. We therefore investigated the use of ASL MRI for the assessment of patients with CKD.

Materials and Methods

Patients with CKD were recruited from the general nephrology clinic at the Glasgow Renal and Transplant Unit, whilst healthy volunteers (HV) were recruited via local advertisement. Subjects attended on a single occasion, undergoing clinical and biochemical assessment, and subsequent MRI. All subjects gave written informed consent and the local ethics committee approved the study. The study is registered with a clinical trials database (ISRCTN 12301736) and was carried out in compliance with the Declaration of Helsinki.

Biochemical measurements

Baseline serum biochemistry and haematology measurements and urinary protein and creatinine quantification were obtained. Estimated glomerular filtration rate (eGFR) was calculated from the measured serum creatinine using the Chronic Kidney Disease Epidemiology Collaboration (CKD–EPI) formula [13]. Proteinuria was measured using a spot protein to creatinine ratio (PCR) from a random urine sample.

Magnetic resonance imaging

MRI was performed on a Siemens Magnetom Verio 3.0 Tesla scanner (Siemens Erlangen, Germany), using a 6-channel phased array body coil. A half Fourier acquisition single shot turbo spin echo (HASTE) localizer sequence was used to identify the location of the kidneys and vessels, using the following parameters: TR = 1400 ms, TE = 93 ms, voxel size = 2.1 x 1.5 x 5 mm3, refocusing pulse flip angle = 160^{o}, number of slices = 30, turbo factor = 179, bandwidth = 781 Hz/pixel. ASL was performed using a flow-sensitive alternating inversion recovery (FAIR) perfusion preparation with true fast imaging and steady precession (True-FISP) acquisition. A single sagittal double oblique slice of both kidneys was obtained, positioned at the mid-point of each axis, moved posteriorly to avoid major vessels. Three images with alternating selective and non-selective inversions were obtained in a single

acquisition during a 12 second breath-hold, and this was repeated five times. In addition, an image with no ASL preparation was acquired to measure equilibrium magnetization. Fair True-FISP parameters were: inversion time 900 ms, repetition time 3.65 ms, echo time 1.83 ms, flip angle 60^{o}, field of view 380 x 380 mm, in plane resolution 2.0 x 1.5 mm^2, matrix size 256 x 192, and slice thickness 10 mm. T1 was acquired during a separate breath-hold by a modified Look-Locker inversion recovery (MOLLI) sequence, with the following parameters: TR = 740 ms, TE = 1.1 ms, voxel size = 2.0 x 1.5 x 6 mm^3, flip angle = 35^{o}, starting inversion time (TI) = 125 ms, TI increment = 80 ms, number of inversions = 3, bandwidth = 930 Hz/pixel. T1 was computed pixel wise using a non-linear curve fitting algorithm, using the three parameter signal model [14]. Total scan time was approximately 15 minutes.

Image analysis

Renal anatomy was assessed on localizer images using a commercially available multi-modality post processing workstation (Siemens Syngo, Siemens, Erlangen, Germany). Kidney length was measured on coronal images and volume was measured using a voxel count method by tracing contours on each slice of a 22 slice transverse oriented image volume. T1 time was measured in cortex, medulla, and whole kidney, and corticomedullary differentiation (CMD) was calculated as the ratio of cortex to medulla T1 time. Post processing was performed using in house software (MATLAB 8.4 R2014b; MathWorks, Natick, Massachusetts, U.S.A). An averaged ASL subtraction image was produced from registered subtraction images derived from each breath-hold. This was fitted to the M0 and T1 data using the standard ASL kinetic model [15] to produce a perfusion map. Image co-registration of ASL, M0 and T1 maps was performed using an enhanced correlation coefficient maximization algorithm with affine transformations [16]. Pixel wise computation of perfusion was performed according to the following formula, where f = perfusion, λ =

tissue-blood partition coefficient (0.8 mL/g in kidney), M0 = equilibrium magnetisation, ΔM = ASL signal, T1 longitudinal relaxation time, TI = inversion time:

$$f = \frac{\lambda}{2TI} \frac{\Delta M(TI)}{M_0} exp \frac{TI}{T1}$$

Regions of interest (ROIs) were drawn onto the perfusion map to measure cortical and whole kidney perfusion. Total single kidney perfusion, was calculated by multiplying the renal perfusion normalised per gram of renal tissue, by the renal mass, assuming that the mass of 1g per $1cm^3$ of renal tissue. For each individual the total kidney perfusion, analogous to renal blood flow was calculated by combining the kidney blood flow of the left and right kidneys. A single operator performed image analysis.

Statistical analysis

Results are expressed as mean ± standard deviation. T1 time and perfusion were measured in cortical and whole kidney ROIs, and ASL measurements are expressed by unit of mass (100g) which is typical in the standard kinetic model. Between group differences in T1 time, CMD, and perfusion were evaluated using independent samples Student's t tests. Evaluation of correlation between MRI measurements and serum and urine biochemistry parameters was performed using Pearson's correlation coefficient. Throughout, p values < 0.05 were deemed significant. Data were analysed using IBM SPSS Statistics version 22.0 (IBM, Armonk, New York, U.S.A).

Results

Baseline data

24 HV and 17 patients with CKD were recruited; the demographic data for each group is displayed in table 1. The CKD group was significantly older ($p < 0.05$), and had higher blood pressure ($p < 0.05$). CKD-EPI eGFR was 39.8 ± 25.2 ml/min/1.73m^2 in the CKD group and 99.6 ± 14.0 ml/min/1.73m^2 in the HV group ($p < 0.001$).

Renal anatomy

Renal anatomical data is shown in table 2. Kidney length was significantly shorter in the CKD group compared with the HV group ($p < 0.05$) however renal volume was no different between the two. The CKD group had significantly higher T1 longitudinal relaxation time both measured in the cortex ($p < 0.001$) and the whole kidney ($p < 0.01$) ROI (figure 1). Furthermore, CMD was significantly higher in CKD than in HV ($p < 0.001$).

Renal perfusion

Renal perfusion was significantly lower in the CKD group (table 2 and figure 1). In the CKD cohort, mean cortical perfusion was 136 ± 37 ml/min/100g in comparison to 279 ± 69 ml/min/100g in the HV cohort ($p < 0.001$). Similarly, whole kidney perfusion was reduced in the CKD group, at 146 ± 24 ml/min/100g compared to 221 ± 38 ml/min/100g ($p < 0.001$). Furthermore, total renal perfusion was 446 ± 150 ml/min in CKD compared to 731 ± 158 ml/min in HV ($p < 0.001$). Typical perfusion maps from both groups are shown in figure 2.

Intra-observer variability

Intra-observer variation of cortical perfusion measurements was 7.3% with intra-class correlation (ICC) of 0.98, whilst variation of whole kidney perfusion measurements was found to be 4.4% with ICC of 0.96.

Association between renal anatomical and functional parameters

There was significant, negative, association between T1 longitudinal relaxation time and ASL perfusion measured in both the cortex ($r = -0.75$, $p < 0.001$) and whole kidney ($r = -0.50$, $p < 0.001$). No significant association was seen between kidney length or volume and either ASL perfusion measurements, or T1 longitudinal relaxation time.

Correlation of clinical, biochemical and MRI parameters

Both cortical and whole kidney perfusion were found to have a negative association with age (respectively, $r = -0.48$, $p < 0.01$; $r = -0.51$, $p < 0.01$). Whilst there was no association between blood pressure and cortical perfusion, a negative correlation was observed between whole kidney perfusion and mean arterial blood pressure ($r = -0.33$, $p < 0.05$).

Correlation was seen between eGFR and both whole kidney T1 longitudinal relaxation time ($r = -0.40$, $p < 0.05$) and cortical T1 time ($r = -0.58$, $p < 0.001$). Furthermore, significant correlation was seen between eGFR and both cortical perfusion ($r = 0.73$, $p < 0.01$) and whole kidney perfusion ($r = 0.69$, $p < 0.01$). There was also significant correlation between total renal perfusion and eGFR ($r = 0.69$, $p < 0.01$). PCR was negatively correlated with both cortical ($r = -0.60$, $p < 0.01$) and whole kidney perfusion ($r = -0.43$, $p < 0.05$) (figure 3).

Discussion

CKD has a tendency to worsen despite treatment of blood pressure and any other reversible or aetiological factors, and there is evidence that common pathological mechanisms are responsible for this irrespective of the original renal insult. Renal damage has been shown to correlate primarily with tubulointerstitial injury [17], characterised by a vicious cycle of microvasculature dysfunction leading to tubular atrophy and fibrosis [18]. In vivo biomarkers to assess renal progression are lacking and emerging techniques such as ASL MRI may provide much needed insight into renal perfusion and thus extent of renal damage.

We found that cortical perfusion is reduced from 279 ml/min/100g in HV to 136 ml/min/100g in patients with CKD, with correlation between perfusion and degree of renal impairment quantified by eGFR. Whole kidney perfusion is similarly reduced, from 221 ml/min/100g to 146 ml/min/100g. This is in keeping with previous measurements of renal perfusion in health and disease, and the finding of reduced native kidney perfusion in CKD has also previously been demonstrated [9,12]. Whilst our perfusion values are lower than found in other studies, this CKD cohort represents the largest to undergo ASL MRI and included patients with more advanced renal impairment than previously studied. Our findings demonstrate strong correlation of renal function to perfusion across a broad range of CKD-EPI eGFR, ranging from 20 to 126 ml/min/1.73m^2.

Earlier human studies using ASL MRI are summarised in table 3, which demonstrates the range of perfusion values previously demonstrated. The broad range could be ascribed to differences in ASL sequence, imaging strategy, and post processing as well as true differences in study population. For example, different strategies have been employed to circumvent the problem of renal respiratory motion, including breath-holding, respiratory gating, or post processing registration. Gardener & Francis [19] found no difference in

perfusion measurements made with either breath-holding or free breathing, but found reduced perfusion when background suppression was used to improve image quality, showing that some variations in imaging approach cause differences in perfusion measurements. Our ASL technique resulted in a scan time of 15 minutes, and breath-holding time of 12 seconds, which was tolerated by all participants.

ASL has been validated in animal models using microsphere techniques and using explanted organs undergoing haemoperfusion. In normal renal function, strong correlation between ASL and both DCE MRI perfusion [20], and PAH clearance [21] has been shown. Validation of ASL against a gold standard perfusion technique has not, to our knowledge, been undertaken in a CKD population. Given that quantitative measurement of perfusion using the standard ASL kinetic model is dependent on T1 time, it is possible that structural changes in CKD are at least partly responsible for the functional changes measured by ASL MRI. In keeping with previous studies [22], we have shown that T1 time is significantly higher in CKD, and that T1 time shows strong correlation with CKD-EPI eGFR. Lee et al [23] previously showed that cortical T1, but not medullary T1 time showed strong correlation with single kidney GFR measured by renography. These differences may be accounted for by changes in extracellular composition, fibrosis, or in the microvasculature, and further investigation is required into the association between the pathological changes in CKD, T1 time, and ASL perfusion. Notably, there was stronger association between eGFR and whole kidney ASL perfusion ($r = 0.69$, $p < 0.01$) than T1 time ($r = 0.40$, $p < 0.05$) suggesting that ASL does grant some additional information into renal physiology in CKD, in addition to the structural changes identified by differences in T1 time. Additionally, there was no association between ASL and kidney size or volume, suggesting that the difference in perfusion in CKD is not entirely attributable to tissue atrophy.

ASL MRI is but one of a number of emerging MRI techniques which may have utility in CKD, such as blood oxygen level dependent (BOLD) imaging, and diffusion weighted (DWI) and diffusion tensor imaging (DTI). Future research should be guided at identifying the imaging correlates of renal fibrosis in CKD, as this may allow identification of biomarkers which can prognosticate, guide therapy, and act as surrogate markers of renal progression in studies of novel therapeutics in CKD.

Our study has a number of limitations. Our CKD cohort has a variety of renal pathologies and whilst common pathological mechanisms underpin all chronic kidney disorders it is possible that perfusion abnormalities may predominate in certain aetiologies of CKD over others. Despite attempts to match the two groups, mean age was higher in the CKD than HV cohort, and therefore the changes in ASL MRI may not be independent of aging. Despite being one of the largest ASL studies in CKD, even larger studies are required to confirm our findings and exclude the possibility of group effects confounding some of the associations with the biochemical parameters which were measured. Furthermore, we have used the standard ASL kinetic model which is primarily validated in healthy volunteers and assumes constant arterial transit time and blood tissue exchange. Differences in these factors may artefactually alter perfusion measurements in CKD, and as previously discussed further research is necessary to validate the use of ASL in the CKD population. Lastly, our study was carried out using 3.0T MRI, which is in general less available in clinical use and further work will be required to translate our findings to 1.5T platform, as it is more commonly used in clinical practice.

In conclusion, we have shown significant differences in renal perfusion measured with ASL MRI in a group of patients with advanced CKD, and shown correlation to renal parameters such as eGFR.

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Conflict of interest statement

There are no conflicts of interest to declare.

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Legends to Figures

Figure 1

Box and whisker plot of T1 longitudinal relaxation time and perfusion in healthy volunteers and CKD.

Figure 2

ASL MRI perfusion maps from a healthy volunteer (A) and patient with chronic kidney disease stage 3/4 (B) with an eGFR of 30 ml/min/1.73m2. Both whole kidney (1 & 2) and cortical (3 & 4) perfusion are demonstrated. Cortical thinning, reduced corticomedullary differentiation, and reduced global perfusion can be seen in CKD.

Figure 3

Association between biochemical measurements and ASL. Correlation was observed between eGFR and cortical perfusion ($r = 0.73$, $p < 0.01$) (A), and eGFR and whole kidney perfusion ($r = 0.69$, $p < 0.01$) (B).

Tables

Table 1. Baseline parameters. Baseline clinical and biochemical parameters are shown of healthy volunteers and patients with chronic kidney disease. Results are shown as mean ± standard deviation.

Parameter	Healthy volunteers	Chronic kidney disease	p value
Number	24	17	
Age (years)	47 ± 14	56 ± 10	< 0.05
Body mass index (kg/m^2)	26.5 ± 5.3	29.3 ± 3.4	0.06
Blood pressure (mmHg)	132/83 ± 15/8	151/90 ± 26/14	< 0.05
Mean arterial blood pressure (mmHg)	99 ± 9	110 ± 17	< 0.05
CKD-EPI eGFR (ml/min/1.73m2)	99.6 ± 14.0	39.9 ± 25.2	< 0.001
Serum creatinine (µmol/L)	68 ± 10	184 ± 69	<0.001
Primary renal diagnosis (number)			
Diabetes		2	
Glomerulonephritis		8	
Renovascular disease		4	
Other		2	
CKD-cause unknown		1	
CKD stage (number)			
1		2	
2		1	
3		4	
4		10	
5		0	

Table 2. MRI parameters. Measurements of renal anatomy, T1 longitudinal relaxation time, and ASL MRI perfusion are shown in healthy volunteers and chronic kidney disease. Results are shown as mean ± standard deviation.

Parameter	Healthy volunteers	Chronic kidney disease	p value
Kidney length (cm)	10.5 ± 0.8	9.7 ± 0.9	< 0.05
Kidney volume (cm^3)	167.1 ± 35.0	160.1 ± 53.4	0.62
Cortical T1 time (ms)	1366 ± 122	1529 ± 77	< 0.001
Whole kidney T1 time (ms)	1472 ± 91	1550 ± 81	< 0.01
Corticomedullary differentiation	0.84 ± 0.07	0.94 ± 0.07	< 0.001
Mean cortical perfusion (ml/min/100g)	279 ± 69	136 ± 37	< 0.001
Mean whole kidney perfusion (ml/min/100g)	221 ± 38	146 ± 24	< 0.001
Mean kidney perfusion (ml/min)	366 ± 79	223 ± 75	< 0.001
Total renal perfusion (ml/min)	731 ± 159	446 ± 150	< 0.001

Table 3. Human studies in arterial spin labeling, grouped by cohort (kidney disease, hypertension, and healthy volunteers). Perfusion measurements are displayed as mean ± standard deviation unless otherwise stated. TI = inversion time, FAIR = flow sensitive inversion recovery, GRASE = gradient and spin echo, FISP = fast imaging with steady state precession, SSFSE = single shot fast spin echo, SSFP = single shot free precession, spin-echo echo planar imaging, BS = background suppression, HASTE = half Fourier acquisition single shot turbo spin echo, UFLARE = ultra-fast low angle rare, T = Tesla, HF = heart failure, HV = healthy volunteer, HTN = hypertensive, CKD = chronic kidney disease, RAS = renal artery stenosis, eGFR = estimated glomerular filtration rate.

Author	Journal	Year	ASL	Field strength	Population & number	Perfusion (ml/min/100g)		
						Whole kidney	Cortex	Medulla
Breidthardt et al [24]	*Eur. Radiol.*	2015	FAIR	1.5T	HF eGFR < 60 ml/min/100g (n=10)		146 ± 50	
					HF eGFR > 60 ml/min/100g (n=10)		171 ± 31	
					Age matched HV (n=10)		274 ± 65	
					HV < 40 years (n=10)		278 ± 59	
Heusch et al [11]	*J. Magn. Reson. Im.*	2013	FAIR True FISP	1.5T & 3T	Kidney transplant			
					eGFR > 30	282.7 ± 60.8		
					eGFR < 30	178.2 ±63.3		
Rossi et al [9]	*Invest. Radiol.*	2012	FAIR True FISP	3T	HV (n=8)	301 ± 51	329 ± 52	
					CKD (mean eGFR 69 ± 12 ml/min by inulin clearance) (n=9)	244 ± 77	263 ± 81	
Artz et al [12]	*Magn. Reson. Imaging.*	2011	FAIR b-SSFP	1.5T	HV (n=5)		427 ± 20	85 ± 33
					CKD (n=5)		225 ± 85	60 ± 23
					Kidney transplant with eGFR > 60 ml/min/1.73m^2 (n=5)		314 ± 41	37 ± 21
					Kidney transplant with eGFR < 60 ml/min/1.73m^2 (n=10)		235 ± 91	36 ± 14

Lanzman et al [10]	*Eur. Radiol.*	2010	FAIR True FISP	1.5T	Kidney transplant with stable function (n=6) Recent kidney transplant (n=7) Acute transplant dysfunction (n=7)		304.8 ± 34.4 296.5 ± 44.1 181.9 ± 53.4	
Fenchel et al [25]	*Radiology*	2006	FAIR True FISP	1.5T	Patients with RAS (n=12)	Asymmetry of perfusion values Significant differences between perfusion in kidney with high grade compared to no or low grade RAS		
					HTN but no RAS (n=6)	243 ± 59	323 ± 79	113 ± 22
Michaely et al [8]	*Invest. Radiol.*	2004	FAIR HASTE	1.5T	CKD (renovascular or other aetiology) (n=46)	Not quantified but reduced ASL signal on semi- quantitative analysis		
Ott et al [26]	*CJASN*	2013	FAIR True FISP	1.5T	HTN, before and after renal denervation (n=19)	256.8 (IQR 241 – 278)		
Schneider et al [27]	*CJASN*	2012	FAIR True FISP	1.5T	HTN, before and after 4 weeks of oral aliskiren therapy (n=34)	272 ±25		
Ritt et al[21]	*NDT*	2010	FAIR True FISP	1.5T	Males with metabolic syndrome before and after 2 weeks of oral telmisartan therapy (n=24)	253 ±20		
Cutajar et al	*Eur. Radiol.*	2014	Multi TI FAIR 3D	1.5 T	HV (n=16)		263± 41	

[20]			GRASE					
Gillis et al [7]	*BMC Nephrol.*	2014	FAIR True FISP	3 T	HV (n=12)	229 ± 41	327 ± 63	
Park et al [28]	*Magn. Reson. Imaging.*	2013	Pseudocontinuous ASL	3T	HV (n=1)		320	
Wang et al [29]	*Acad. Radiol.*	2012	FAIR SSFSE	3T	HV, before and after intravenous furosemide (n=11)		366.6 ± 41.2	118.59
Cutajar et al [6]	*MAGMA*	2012	Multi TI FAIR 3D GRASE	1.5T	HV (n=20)	147 ± 30.8	178 ± 40.7	
Gardener AG & Francis ST [19]	*Magn. Reson. Imaging.*	2010	FAIR True FISP SE-EPI with & without BS	1.5T	HV (n=9) FAIR TRUE FISP SE-EPI with BS SE EPI without BS		 367 ± 50 284 ± 75 334 ± 65	 103 ± 27 139 ± 55 122 ± 48
Kiefer et al [30]	*Acad. Radiol.*	2009	FAIR TrueFISP	3T	HV (n=11)		245 ± 11	109 ± 5
Karger et al [31]	*Magn. Reson. Imaging.*	2000	FAIR UFLARE	1.5T	HV (n=10)	213 ± 55		

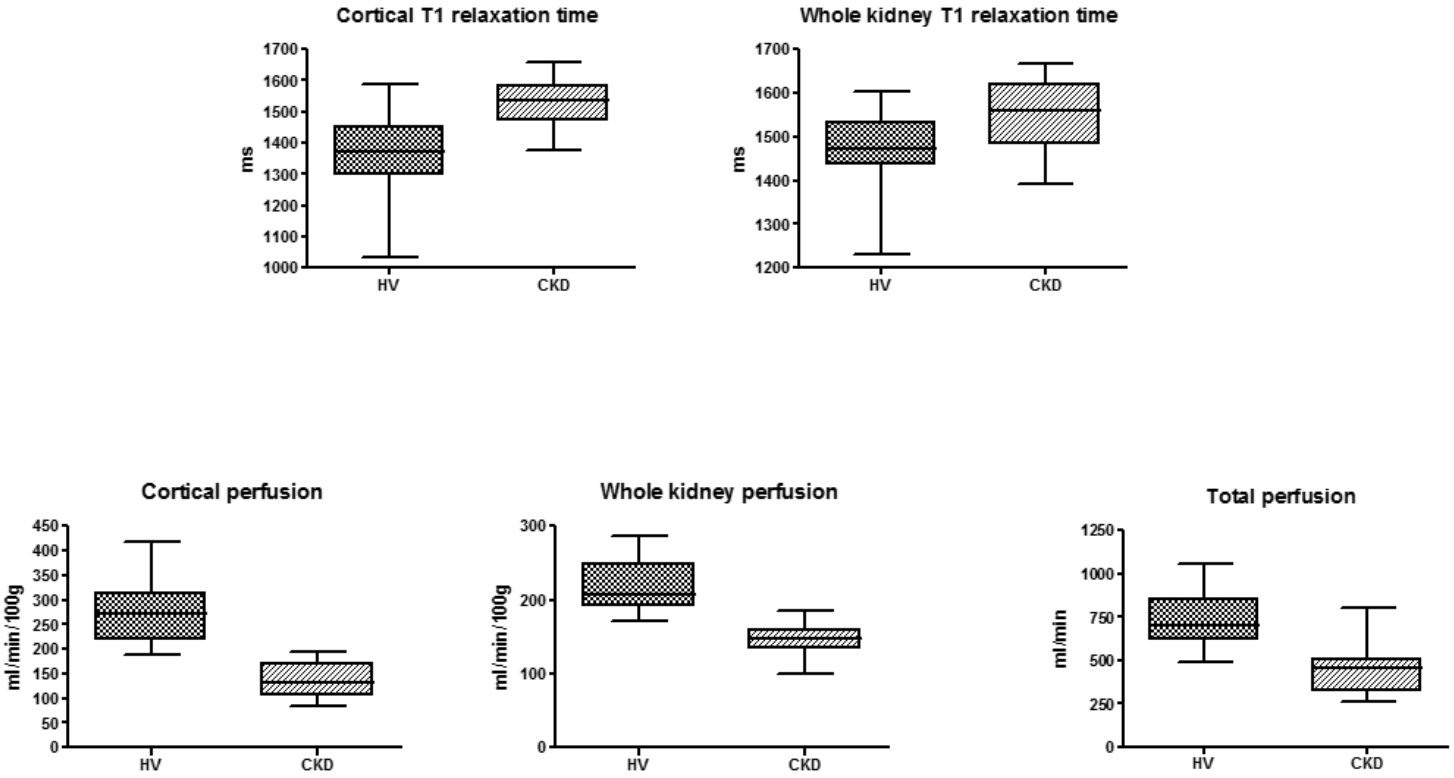
Cortical T1 relaxation time
ms
1700
1600
1500
1400
1300
1200
1100
1000
HV
CKD
Whole kidney T1 relaxation time
ms
1700
1600
1500
1400
1300
1200
HV
CKD
Cortical perfusion
ml/min/100g
450
400
350
300
250
200
150
100
50
0
HV
CKD
Whole kidney perfusion
ml/min/100g
300
200
100
0
HV
CKD
Total perfusion
ml/min
1250
1000
750
500
250
0
HV
CKD

Figure 1

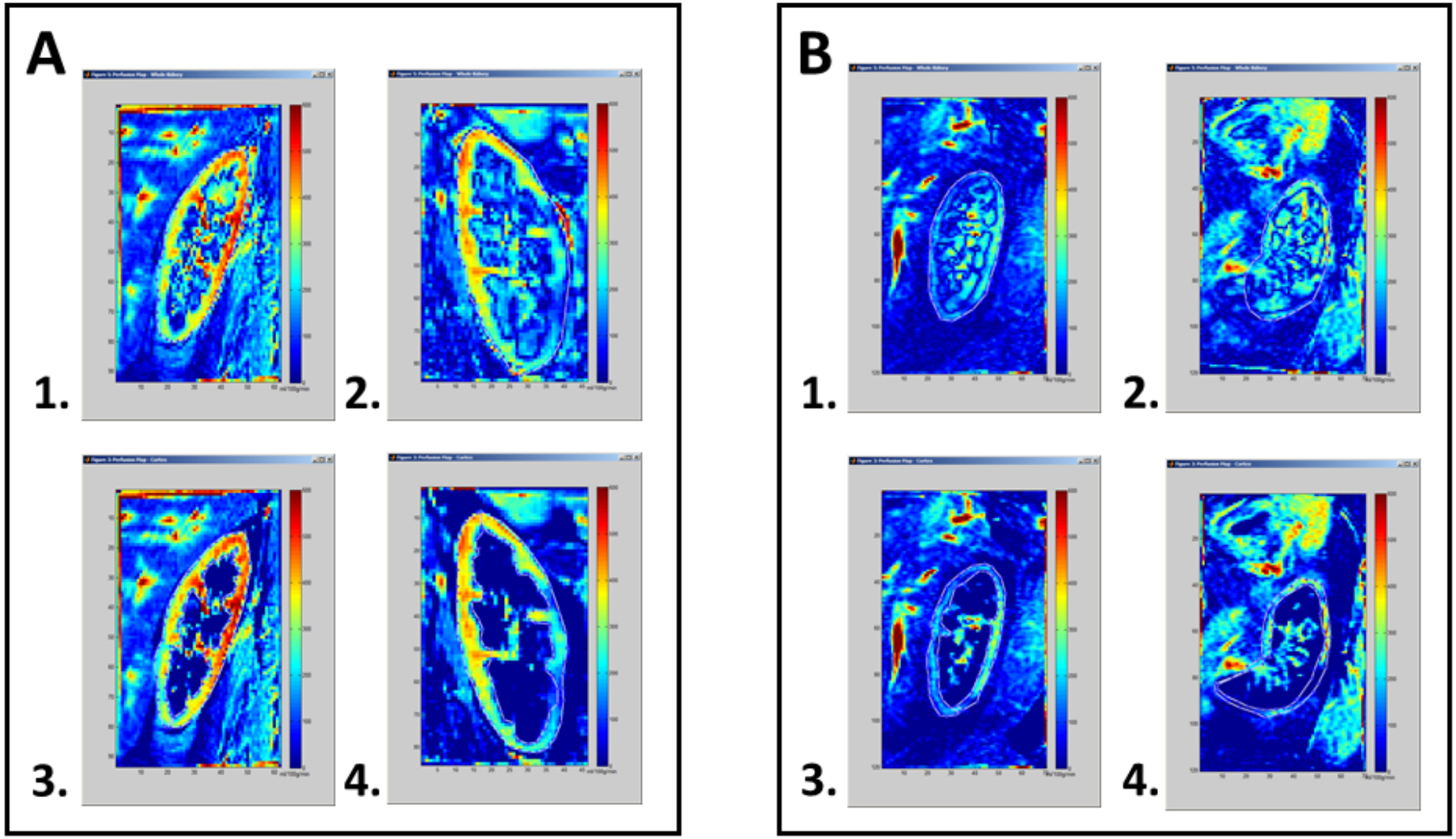

Figure 2

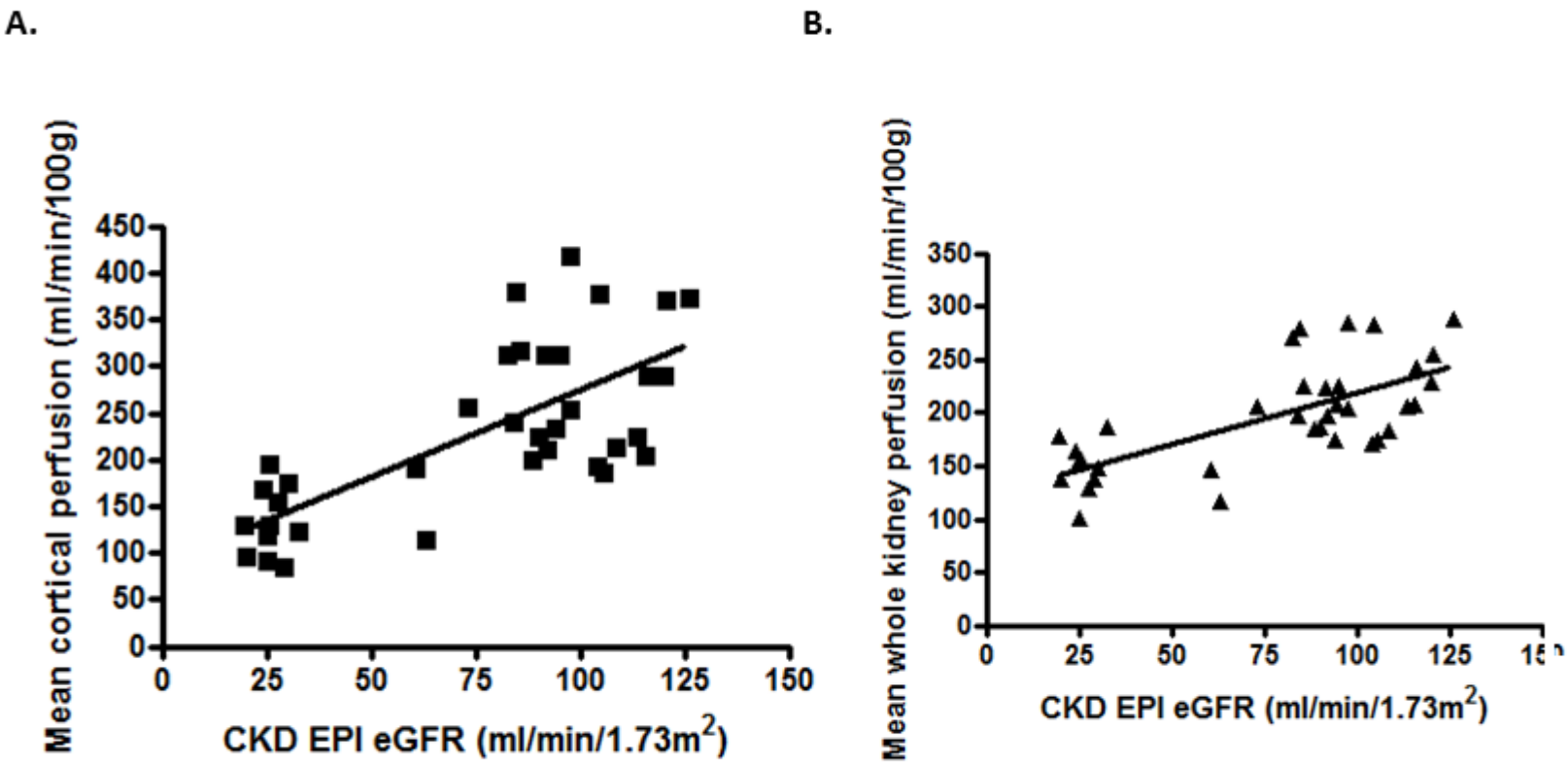
A.
Mean cortical perfusion (ml/min/100g)
450
400
350
300
250
200
150
100
50
0
0
25
50
75
100
125
150
CKD EPI eGFR (ml/min/1.73m2)
B.
Mean whole kidney perfusion (ml/min/100g)
350
300
250
200
150
100
50
0
0
25
50
75
100
125
CKD EPI eGFR (ml/min/1.73m2)

Figure 3