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Title:
Will advances in functional renal MRI translate to the nephrology clinic?

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Abstract (217 words)

Characterizing structural and tissue abnormalities of the kidney is fundamental to understanding kidney disease. Functional multi-parametric renal magnetic resonance imaging (MRI) is a noninvasive imaging strategy whereby several sequences are employed within a single session to quantify renal perfusion, tissue oxygenation, fibrosis, inflammation, and oedema without using ionizing radiation. In this review, we discuss evidence surrounding its use in several clinical settings including acute kidney injury, chronic kidney disease, hypertension, polycystic kidney disease and around renal transplantation. Kidney size on MRI is already a validated measure for making therapeutic decisions in the setting of polycystic kidney disease. Functional MRI sequences, T1 mapping and apparent diffusion coefficient, can non-invasively quantify interstitial fibrosis and so may have a near-future role in the nephrology clinic to stratify the risk of progressive chronic kidney disease or transplant dysfunction. Beyond this, multi-parametric MRI may be utilised diagnostically, for example differentiating inflammatory versus ischaemic causes of renal dysfunction, but this remains to be proven. Changes in MRI properties of kidney parenchyma may be useful surrogate markers to use as end points in clinical trials to assess if drugs prevent renal fibrosis or alter kidney perfusion. Large, multi-centre studies of functional renal MRI are ongoing which aim to provide definitive answers as to its role in the management of patients with renal dysfunction.
Introduction

Over the past four decades the widespread application of diagnostic medical imaging has revolutionised the practice of modern medicine (1,2). However, the functional unit of the kidney, the nephron, is microscopic, and as such, the role of conventional imaging in the diagnosis of kidney disease is limited. Kidney imaging is still vital in the assessment of patients with kidney disease although its application is rudimentary and aims to answer very specific questions which differentiate only a minority of patients. Kidney size and shape, presence or absence of hydronephrosis and perfusion of the kidney are the key questions addressed, most of which can be reliably answered using simple imaging techniques (ultrasound, non-contrast computed tomography) and have not advanced despite major technological innovations over the past 20 years. While there have been advances in techniques to assess large vessel kidney perfusion, their application to the management of many aspects of chronic kidney disease remains uncertain (3). The dearth of imaging applications in nephrology may signal a missed opportunity to improve our diagnostic accuracy and to allow better risk stratification of patients. In Europe, between 10-30% of incident dialysis patients have kidney disease of uncertain aetiology (4). Given that incident dialysis patients represent the most severe ‘tip’ of the kidney disease ‘iceberg’ there are millions of people worldwide with life-limiting kidney disease who do not have an accurate diagnosis. Diagnostic tools in nephrology in routine clinical care are limited. Creatinine, glomerular filtration rate (estimated or measured), cystatin C and proteinuria are non-specific markers and poorly differentiate the aetiologies of kidney disease (5). Proportionately few kidney diseases have specific serological biomarkers (6), and although serological markers of renal disease are continuously expanding (7), most patients with positive serology still require confirmatory histology. As such, kidney biopsy remains the gold standard diagnostic tool (5). However, a kidney biopsy is an invasive procedure which risks life-threatening bleeding (8), has several relative complications and is vulnerable to
sampling error. Accordingly, there is a need for novel, non-invasive biomarkers of kidney disease to reduce risk exposure and increase diagnostic accuracy in kidney disease. While advances in exome sequencing is likely to identify previously unknown actionable genetic diagnoses in just under 10% of the dialysis population, the nephology community should embrace as many state of the art advances in diagnostic technologies to advance application of precision medicine to patients with kidney disease.

Functional magnetic resonance imaging (MRI) of the kidney is a burgeoning area of interest where it is hoped that different MRI sequences can provide quantitative information regarding perfusion, tissue oxygenation, fibrosis, inflammation, and oedema, in addition to the standard anatomical and structural assessment that 3D imaging allows. Where these different sequences are acquired in a single sitting, typically lasting 30-60 minutes, it is referred to as ‘multi-parametric’ MRI.

**MRI sequences of interest**

Functional renal MRI can be performed on 1.5T or 3T MRI scanners, where T refers to tesla, the unit of magnetic field strength. Most clinical MRI scanners are 1.5T, and although, in theory, the stronger the magnet the greater the achievable image resolution, this is not always true in practice. Standard contraindications to MRI apply to renal MRI and are becoming less restrictive with most implanted cardiac devices now deemed MRI conditional. Detailed description of each MRI sequence is beyond the scope of this review, but Table 1 summarises the most commonly acquired MRI sequences that are combined into a multiparametric protocol: diffusion-weighted imaging (DWI) using apparent diffusion coefficient (ADC), T1 mapping, blood oxygen level dependent imaging (BOLD) and arterial spin labelling (ASL). These sequences do not require gadolinium based contrast, which is associated with a small risk of nephrogenic systemic fibrosis.
following in patients with advanced kidney disease (22,23). Gadolinium based contrast agents are still critical for certain techniques such as contrast-enhanced magnetic resonance angiography (CE-MRA) and dynamic contrast enhanced (DCE) renal perfusion. Future renal MRI may adopt non-traditional MRI contrast agents including iron-based agents such as ferumoxytol (24,25). Supplementary material table S1 outlines a non-exhaustive list of additional sequences that may be included in imaging protocols relevant to functional renal MRI.

Clinical Applications of Renal MRI

Polycystic Kidney disease
Total kidney volume (TKV) measured on MRI is a validated prognostic marker in patients with polycystic kidney disease, such that the Food and Drug Administration and the European Medicines Agency have both approved height-adjusted TKV to guide treatment decisions in patients with polycystic kidney disease (26). This follows the seminal findings of the CRISP study, a longitudinal study of adult patients with polycystic kidneys which characterised the relationship between total kidney volume (TKV) and measured glomerular filtration rate and found that increasing TKV (resulting from gradual expansion of renal cysts) occurs prior to decline in eGFR and can predict future decline (27). These findings were confirmed by the PKD Outcomes Consortium (28), and featured in the inclusion criteria for the landmark trials showing that tolvaptan reduced the rate of eGFR decline in patients with PKD (TEMPO 3:4 (29) and REPRISE (30)). Height-adjusted TKV can also be combined with age to determine The Mayo Imaging Class, which has been shown to successfully distinguish patients at differing rates of progression (31). TKV is most accurately measured from cross-sectional images obtained by MRI or computed tomography using manual segmentation or stereology (where a square grid is super-imposed on each kidney slice before the number of grids containing kidney tissue are manually...
counted). These processes are time-consuming but it is plausible that effortless TKV measurement will soon be possible using machine learning software (32). In the meantime, an alternative method using an ellipsoid equation, where coronal, sagittal and transverse diameters are used to estimate TKV have also been shown to be acceptable for majority of cases (31). Outside the setting of PKD, reducing kidney volume has been shown to correlate with reduced eGFR, including in the setting of living kidney donors (33,34).

**Acute Kidney Injury**

In a minority of patients with acute kidney injury (AKI), anatomical MRI sequences may support a specific diagnosis, for instance, there are well described appearances of renal cortical necrosis evident on T2-weighted images and contrast-enhanced T1 images (35,36). Similarly, specific patterns suggesting the sequelae of acute tubular necrosis or presence of pyelonephritis may be detected (37). Multi-parametric functional renal MRI has not been extensively studied in patients with non-transplant acute kidney injury (AKI). The only study of multi-parametric renal MRI in AKI included follow-up data for only seven participants (38). Nevertheless, this study identified significant increases in TKV, T1 relaxation time, and cortical perfusion that gradually improved during recovery from AKI. Interestingly, by three months serum creatinine returned to normal in all subjects, but the MRI parameters remained abnormal in some. Additional small studies focusing on single MRI sequences have also shown that perfusion measured by ASL and phase-contrast MRI was reduced in patients with AKI compared to healthy controls (39,40), while studies examining BOLD found no difference between AKI and healthy volunteers (although changes were evident in healthy volunteers after the administration of furosemide that did not occur in those with AKI) (41). Inoue *et al.* examined BOLD and ADC in a sub-group of 23 patients with AKI and found no correlation between either parameter and estimated glomerular filtration rate (eGFR) (42). This is not surprising given the
limited relevance of eGFR in AKI. Raman et al. found increased cortical T1 in 52 patients with COVID-19 (of whom 6 had AKI) compared to 28 controls (43).

MRI in AKI can be logistically challenging due to the need for participants with an acute illness to lie flat for up to one hour and co-ordinate multiple breath-holds (38). It is likely that technological advances in shortened acquisition times (through the use of methods such as compressed sensing) and free-breathing sequences will reduce this burden (44).

**Clinical application of MRI in AKI:**
- Unclear, potentially limited due to fluctuant clinical status and burden to patient.
- Future studies should address if functional MRI can distinguish AKI of haemodynamic versus inflammatory aetiology
- Serial scanning could guide prognostication of renal recovery in AKI, but this remains to be proven.

**Chronic Kidney Disease**
Reduction in kidney volume and loss of corticomedullary differentiation are established, non-specific markers of chronic kidney disease (CKD) that have been diagnosed using T1 weighted MRI images for over 30 years (45–47). Beyond anatomical assessment, there are numerous studies examining the role of functional MRI in patients with CKD. Inoue et al. examined BOLD and DWI in 119 patients with CKD and found ADC measured from the DWI sequence correlated with eGFR ($r^2 = 0.56$ in patients with diabetes, and 0.46 in non-diabetic CKD). In a subgroup of 37 patients without diabetes who underwent kidney biopsy, they found a significant correlation between the degree of fibrosis with ADC ($r^2 = 0.48$, $p<0.01$) and BOLD ($r^2$...
Another prospective study, this time of 112 patients with CKD and 71 controls found that cortical oxygenation measured as R2* on BOLD, negatively correlated with eGFR, with those individuals with higher cortical values being three times more likely to develop a need for renal replacement therapy or a 30% increase in serum creatinine (48). Zhang et al. studied MR elastography and DWI in 97 adults with CKD to find the former negatively correlated with histological fibrosis (ρ = -0.397), while the latter positively correlated (ρ = 0.472) with the density of peritubular capillaries (a surrogate for fibrosis) (49). Two key studies adopted a multi-parametric approach. Buchanan et al. studied 44 patients without diabetes who had CKD (50). They found that multiparametric MRI was able to differentiate CKD from controls with normal kidney function (CKD values included higher T1, lower ADC, lower ASL), with correlation between MRI and clinical parameters (eGFR and urine protein:creatinine ratio). MRI parameters (T1 and ADC [both cortical values and cortico-medullary differentiation], as well as cortical perfusion on ASL), correlated with histology and were able to differentiate high versus low levels of fibrosis on histology with an optimal threshold of 40% fibrosis. Berchtold et al. used a multiparametric protocol in a study of 164 patients with CKD, although the majority of these were transplant recipients (51). In the 46 participants with native kidney CKD, the difference in cortex-to-medulla (Δ) ADC and ΔT1 values correlated with the % fibrosis r = 0.64, p < 0.001 and r = 0.71, p < 0.001 respectively. The association was still present, although weaker, when cortical values were used. Across the entire cohort (native and transplant kidneys), combining ΔADC, ΔT1 and eGFR into a multivariable model successfully distinguished patients with high and low levels of fibrosis. The model could identify patients with minimal fibrosis (<10%) with a positive predictive value (PPV) 95.5%, and those with high levels of fibrosis (>50%) with PPV = 88.8%. Perhaps most encouragingly there was no interaction between ΔADC, ΔT1 and eGFR, and model fit increased from r² = 0.30 for eGFR alone to 0.54 when the MRI
parameters were added. There are two ongoing, large scale multi-centre studies. The Prognostic Imaging Biomarkers for Diabetic Kidney Disease (iBEAt) study looks to recruit 500 participants with diabetic kidney disease and perform serial scanning over 3 years to identify prognostic MRI biomarkers (ClinicalTrials.gov Identifier: NCT03716401). The Application of Functional Renal MRI to Improve Assessment of Chronic Kidney Disease (AFiRM) study will recruit 450 participants to examine if multi-parametric renal MRI can characterise patients with and without CKD progression (ClinicalTrials.gov Identifier: NCT04238299). This study includes a mechanistic sub-study of 45 participants which aims to compare MRI findings with histology.

**Clinical Application of MRI in CKD:**

- The near-future clinical application of multi-parametric functional MRI in patients with CKD is the non-invasive quantification of fibrosis to inform prognostication and guide decisions regarding biopsy and management.
- MRI will also be able to inform if fibrosis on a kidney biopsy sample is representative of the organ as a whole
- Renal MRI may have a role as a surrogate outcome measure in future trials of clinical therapeutics
- It may be possible to use MRI to stratify which patients gain most benefit from future therapeutic strategies.
- A large multi-centre study is ongoing to assess if multiparametric MRI can distinguish patients with CKD who have progressive versus stable disease

**Hypertension**

Hypertension is the second leading cause of renal failure in the Western world (52) and MRI may disclose both causes of raised blood pressure and the effects of it (37). The commonest cause of secondary hypertension is renal parenchymal disease
(which may be multifactorial) while arteriosclerotic or fibromuscular renovascular
disease and obstructive uropathy are also important causes. This especially so
given that angioplasty for renal artery fibromuscular dysplasia or mechanical relief of
hydronephrosis may effect a complete cure and be extremely cost-effective in the
individual, negating the expense of many years of drug therapy, associated
monitoring costs and potential drug side-effects.

For renovascular imaging magnetic resonance angiography (MRA) is key to
providing an accurate depiction of the arteries and given the variability of anatomy
and high spatial resolution required, contrast-enhanced (CE-MRA) techniques are
preferred (figure 1) (53,54). The protocol can also integrate dynamic contrast
evaluation to evaluate differential perfusion (55,56). Since the adjacent adrenal
glands will also be imaged then adrenal secondary causes of hypertension in the
form of primary aldosteronism (e.g. Conn’s tumour) and phaeochromocytoma may
be disclosed.

**Kidney Transplant Recipient Imaging**
Renal MRI has been studied actively in renal transplant recipients, largely driven by a
very clear clinical question: can MRI distinguish rejection from other causes of
transplant dysfunction? Most of these studies had small sample sizes and focused
on a single MRI sequence with conflicting results (57). At present, it is not clear if any
parameter, alone or in conjunction, can reliably distinguish causes of allograft
dysfunction. Moreover, given the advances in urinary and serum biomarkers for
acute rejection it is plausible that non-imaging techniques may fulfil this clinical
need. (58,59) The ability of MRI to detect fibrosis in allografts has yielded more
consistent and promising results. In addition to the study discussed above by
Berchtold *et al.* (which included 118 participants with renal transplants) (51), there
are 3 notable studies comparing multi-parametric renal MRI with renal histology.
Friedli et al. report MRI findings in 33 renal transplant recipients undergoing clinically indicated transplant kidney biopsy. They found that ADC and T1 were not sensitive for interstitial inflammation. However, ΔADC (defined as cortex minus medullary values) negatively correlated with histological fibrosis ($r^2 = 0.64$, p<0.001), such that a negative ΔADC yielded 100% sensitivity and 71% specificity to discriminate fibrosis of 40% or more. Wang et al. performed multi-parametric MRI in 103 renal transplant recipients who were undergoing clinically indicated biopsies (60). Again, ADC was found to negatively correlate with interstitial fibrosis ($\rho = 0.77$, p<0.001), with ASL and BOLD also significantly associated with fibrosis ($r = 0.77$, p<0.001 and $\rho = 0.61$, p <0.001, respectively). Each variable was able to discriminate patients with and without 50% fibrosis with an area under the curve of ≥0.85. Bane et al. compared MRI findings in 27 renal transplant recipients in whom 15 had stable allograft function and 12 had chronic dysfunction (eGFR <30 ml/min/1.73 m$^2$). Once again, ADC and ΔT1 differentiated the functioning allografts from the fibrotic ones, with excellent cross-validated diagnostic performance when used in combination. They also found that cortical ADC and T1 had good performance at predicting an eGFR decline of ≥4 ml/min/1.73 m$^2$ per year at 18 months.

Magnetic resonance elastography, a technique that uses external mechanical vibration to quantify organ stiffness on MRI, is particularly suited to renal transplant recipients given allografts are placed much closer to the skin surface than native kidneys. However, there have been some divergent results in the studies so far (with one study (61) showing a positive correlation between elastography and fibrosis, while another found a negative correlation (62). The technique is also limited by the requirement for the vibration device which is not in routine clinical use.
The other indication for MRI in renal transplant recipients is where renal transplant artery stenosis is suspected, for example with the development of hypertension. CE-MRA allows imaging of a tortuous vascular anatomy to determine if a stenosis has developed and allowing a 3D road-map for any planned endovascular intervention.

**Clinical application of MRI in renal transplant dysfunction:**
- Functional MRI utilising T1 and ADC can reliably detect fibrosis in renal transplants. MRI may have a clinical role in discriminating transplant kidneys with extensive versus minimal fibrosis.
- The role of functional MRI in distinguishing causes of acute transplant dysfunction remains to be proven.

**Potential Kidney Donor Imaging**

Potential kidney donors require comprehensive imaging as part of their assessment to detect any incidental intrinsic renal disease or anatomical variants (particularly the vasculature) that would preclude donation. These altruistic donors are often young and merit a ‘least harm’ approach hence MRI, with its lack of ionising radiation, is preferred to multiphase CT. Given that the vasculature is the most fundamental part of this evaluation, CE-MRA is key to providing high spatial resolution depiction of the arteries and veins although a full imaging protocol can also include dynamic contrast evaluation to evaluate differential perfusion and an excretory urographic phase to image the pelvicalyceal systems and ureters (figure 1).

**Barriers to the Clinical Application of Functional Renal MRI**

MRI will always be limited by cost, availability, and contraindications in some patients (12,13). However, the clinical need for improved diagnostic tools and the burden of
existing techniques is sufficient to justify its inclusion into clinical practice if its utility is proven. As relatively new techniques, the functional MRI approaches also require standardisation of acquisition and validation of analysis methods, which must be widely-available and applicable across MRI vendors and clinical sites. This is being actively addressed by an international collaboration (63). Caution is needed when interpreting the results of studies comparing renal MRI findings to eGFR. For renal MRI to be useful in clinical practice it must offer information over and above that which can be more readily obtained from a routine blood test. Comparing MRI findings to histological fibrosis is more appealing and is both complimentary to renal biopsy and allows serial imaging. Interstitial fibrosis has been shown to be a key prognostic marker in CKD (64). Being able to confidently diagnose interstitial fibrosis without the need for invasive procedures could have major impact on the management and prognostication of a sub-group patients attending the renal clinic. Caution must also be applied when interpreting results that use corticomedullary differentiation (i.e. $\Delta$T1, $\Delta$ADC). The reason for this is two-fold. Firstly, there is not a standardised method for comparing cortico-medullary differentiation, with some papers subtracting medullary values from cortical values, while others use a ratio (cortex divided by medulla) (65). The impact of this on results is likely to be small. Secondly, and more importantly, there is a risk of over interpreting the significance of cortico-medullary findings. Loss of cortico-medullary differentiation is an established, but non-specific, finding in CKD that is detectable on ultrasound, computed tomography and MRI (46). Any observed association between renal outcomes and $\Delta$T1 or $\Delta$ADC may under-utilise MRI as a functional measurement and may instead detect a crude structural change that is prevalent in CKD, and which can be measured in simpler ways. Nevertheless, there is now sufficient data to suggest that ADC and T1 can be used to reliably detect renal fibrosis. Future studies (including
one that is already underway (ClinicalTrials.gov Identifier: NCT04238299)) should now look at the clinical utility of these parameters in addition to routine clinical care.

**Conclusion**

Multi-parametric functional MRI is an exciting area of research. In the near future it is expected that T1 and ADC measurements derived from MRI will be used to reliably and non-invasively quantify interstitial fibrosis in the renal clinic. While it is conceivable that the ultimate role of functional renal MRI will extend beyond this (for instance, differentiating inflammatory versus ischaemic causes of renal dysfunction) this is yet to be proven. Large, multi-centre studies of functional renal MRI techniques are ongoing which will hopefully provide definitive answers as to their role in the management of patients with renal dysfunction.

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**Conflicts of interest**

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Berchtold L, Friedli I, Crowe LA et al. Validation of the corticomedullary


64. Srivastava A, Palsson R, Kaze AD et al. The Prognostic Value of

Table 1: The main MRI sequences included in a multiparametric renal MRI protocol. Representative images acquired using 3T MRI.

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<th>MRI Parameter</th>
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<th>Explanation</th>
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<td>Anatomical images</td>
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<td>Standard anatomical images. These are rapidly acquired in coronal, sagittal and transverse planes over a series of breath-holds. They allow standard clinical structural assessment (e.g. excluding hydronephrosis), measure renal volume (if sufficient slices are taken) and can be used to plan subsequent functional sequences.</td>
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Diffusion weighted imaging (DWI) (14,15)

ADC map. The diffusibility of water within an individual voxel is quantified by the apparent diffusion coefficient (ADC) (mm²/s). This is therefore reduced by fibrosis and inflammation, as well as the presence of microstructures such as glomeruli and capillaries.
T1 mapping (16,17)  

T1 map. T1 (ms) represents the time constant for the recovery of longitudinal re-magnetisation of protons following a pulsed MRI signal. T1 is longer for increased water content. T1 time increases in the presence of fibrosis, inflammation and oedema.
Blood oxygen level dependent imaging (BOLD) (18,19)

T2* map. T2* refers to the observed (rather than natural (T2)) transverse relaxation time constant on MRI (ms) which is affected by magnetic inhomogeneities in the tissue. Deoxyhaemoglobin shortens T2*, and so T2* (or R2*, which is 1/T2*) mapping produced by BOLD imaging reflects local tissue oxygen bioavailability.
| Arterial spin labelling (ASL) (20,21) | Measures renal perfusion (ml/100g/min) without exogenous tracer using magnetically labelled protons in blood. |
Figure 1. Representative images of contrast-enhanced magnetic resonance angiography showing normal renal vasculature including accessory left renal artery (A) and an example right renal artery stenosis due to fibromuscular dysplasia - fibrointimal variant (B). With appropriate modification of the MRI protocol, it is also possible to evaluate regional renal perfusion using dynamic contrast enhanced (DCE) (C) and excretory phase urography (D).
Supplementary Material table S1. Non-exhaustive list of additional MRI sequences that are relevant to renal MRI