TARGET AUDIENCE: Investigators studying kidney disease with MRI

INTRODUCTION:
A recent study in a mouse model of diabetes has used MRI with spectrally selective excitation to show increase in cortical fat in diabetic kidneys compared to normal controls(1). This study raises the possibility that quantification of renal fat might be useful to assess chronic kidney disease severity in humans. We retrospectively evaluated the fat content of kidneys in 14 human subjects with varying degrees of renal failure ranging from normal renal function to dialysis dependence, based on in- and out-of-phase T1-weighted axial images of the kidneys. Correlation between fat content measured with the in-phase/out-of-phase images and estimated glomerular filtration rate (eGFR) or the body mass index (BMI) was investigated.

METHODS:
In-phase and out-of-phase T1-weighted images were obtained through the kidneys of 14 subjects (ages 43 to 82, 9 diabetic) in an IRB-approved study. A central kidney slice was chosen and a region-of-interest (ROI) was drawn on that slice around the kidney and around potential cortical regions. Corticomedullary differentiation on T1-weighted images decreases with severity of renal disease(2). When not clear, small regions were selected near the kidney wall for likely cortical regions. The mean of the fat index ([In-phase] – [Out-of-phase])/[In-phase] (defined as FAT) within the ROI was plotted against eGFR and BMI and the correlation was calculated.

RESULTS:
Cortical FAT ranged from 0.03 to 0.18 in subjects who had an eGFR below 44. We found no correlation between any of the MR-derived fat indices and eGFR (Fig. 2A,B). We also found no significant correlation between fat indices and BMI (Fig. 2C,D) The R² correlation obtained for FAT versus eGFR was 0.04 when observed for the entire kidney and 0.14 for cortical ROIs. The R² correlation obtained for FAT versus BMI was 0.15 when observed for the entire kidney and 0.27 for cortical ROIs.

DISCUSSION:
Renal fat estimated with in- and out-of-phase images does not correlate with eGFR or BMI in this retrospective study. Better methods for fat quantification exist, including multi-point Dixon or IDEAL methods(3). Prospective application of these methods may show subtle changes in renal fat with worsening renal disease. However, the results of our current study suggest that such differences may be slight, if they exist. The results of this study do not disprove the diabetic mouse model(1) as it did not look specifically at diabetic patients. However, this work suggests that the assessment of cortical fat will not indicate general renal dysfunction. Whether diabetic nephropathy results in increased renal fat remains to be assessed.

CONCLUSION:
We found no increase in fat content in the kidneys (estimated by in-phase and out-of-phase images) with renal dysfunction across a range of etiologies.

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