## P0254 MYOCARDIAL TISSUE CHARACTERIZATION IN LIVING KIDNEY DONORS 5 YEARS AFTER NEPHRECTOMY

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**Background and Aims:** Uraemic cardiomyopathy is characterized by left ventricular (LV) hypertrophy, diastolic dysfunction and myocardial fibrosis. Diffuse interstitial fibrosis of the myocardium, assessed using cardiac magnetic resonance imaging (CMR) techniques of native T1-mapping and extracellular volume (ECV), has been demonstrated in end stage chronic kidney disease (CKD) and in patients with stage 3 CKD and normal left ventricular mass. In living kidney donors estimated glomerular filtration rate (eGFR) declines by a third after donation, with 60% having a resultant eGFR comparable with stage 3 CKD. Recent studies have demonstrated small increases in LV mass at 12 months after donation as well as functional sequelae of reduced global circumferential strain and apical torsion. We sought to establish whether living kidney donors had CMR evidence of diffuse interstitial cardiac fibrosis which might contribute to observed functional correlates.

**Method:** A cross sectional blinded study of living kidney donors (n=50) and healthy controls (n=45) who underwent 3 Tesla CMR and blood samples for biomarkers of fibrosis. A modified look-locker inversion recovery (MOLLI) 5(3)3 sampling scheme was used for T1 mapping followed by T2 mapping sequences at the mid LV slice. Five minutes after the administration of gadolinium (Gadovist®) contrast (0.15mmol/kg), standard T1-weighted gradient echo inversion recovery images were repeated for the assessment of late gadolinium enhancement (assessment for focal fibrosis). Post contrast MOLLI images were acquired using identical slice positions as native images using a 4(1)3(1)2 sampling scheme 15 minutes after the administration of gadolinium. Native and post contrast T1 time was used to calculate ECV.

**Results:** There were no differences in demographics between groups; age (donors 54 ± 12yrs vs. healthy controls 50 ± 13yrs, p=0.128), ethnicity (89% Caucasian) or male gender (37%). LV mass and volumes were not significantly different. Forty four donors and 34 controls consented to receive gadolinium contrast. Native T1 in the septal mid LV slice was not significantly different between groups (donors 1223 ± 35ms vs. controls 1210 ± 36ms, p=0.102). There was also no difference in T2 time of the septal mid LV slice (donors 40 ± 2ms vs. controls 40 ± 4ms vs. p=0.455).

Late gadolinium enhancement was seen in five living kidney donors in a right ventricular insertion point pattern but there was no mid wall or ischaemic pattern enhancement. There was no difference in septal ECV at the mid LV slice (donors 25 ± 2% vs. controls 25 ± 2%, p=0.896). There was also no corresponding difference in fibroblast growth factor-23 (RU/ml) in donors 74 [58-105] vs. controls 59 [47-75], p=0.081 or soluble α-klotho (pg/ml) in donors 610 [503-810] vs. controls 703 [550-955], p=0.061.

**Conclusion:** Septal T1 times and ECV in living kidney donors at 5 years from donation are no different from healthy controls. Biomarkers of cardiac fibrosis are also comparable to healthy controls in this small cohort. We found no CMR evidence of the ultrastructural changes reported in uraemic cardiomyopathy in the hearts of living kidney donors at 5 years from donation.