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CARDIOVASCULAR EFFECTS OF UNILATERAL NEPHRECTOMY IN LIVING KIDNEY DONORS AT FIVE YEARS

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Background and Aims: Chronic kidney disease (CKD) is a highly prevalent risk factor for cardiovascular disease with an inverse relationship between estimated glomerular filtration rate (eGFR) and increases in all-cause and cardiovascular mortality. Living kidney donation provides a model to study the cardiovascular effects of a reduced kidney function in previously healthy subjects without comorbidities. We report follow up results in a group of kidney donors and healthy controls who underwent cardiovascular assessment at baseline, 12 months and 5 years after nephrectomy in the CRIB-DONOR studies (NCT01028703, NCT02973607).

Method: A longitudinal blinded end point study of kidney donors (n=50) and healthy controls (n=45) followed up between May 2017 to May 2019. Participants underwent a cardiac MRI (3.0 Tesla) for assessment of left ventricular (LV) size, mass, systolic function (ejection fraction and 3-dimensional feature tracking) and aortic distensibility. Clinical assessment included; office and 24-hr ambulatory blood pressure measures, measurement of arterial stiffness (SphygmoCor) and blood and urine analysis.

Results: Mean follow up time was 5.7 ± 0.7 yrs. Mean eGFR in donors was 95 ± 15 ml/min/1.73m² prior to donation, 65 ± 13 ml/min/1.73m² at 12 months and 67 ± 14 ml/min/1.73m² at 5 years. This compared with an annual decline in eGFR of -1ml/min/1.73m² in healthy controls. Despite a rise in LV mass at 12 months in the original study, by 5 years, LV mass in donors was no different to controls $(113\pm31g$ vs. $115\pm30g$, p=0.707). There was also no significant difference in LV volumes or LV geometry. At 5 years, 3D global longitudinal strain (donors -16.2 $\pm2.5\%$ vs. controls -14.9 $\pm2.1\%$, p=0.007) and 3D global circumferential strain (donors -19.0 $\pm2.5\%$ vs. controls -17.7 $\pm2.2\%$, p=0.004) were marginally greater in donors than controls.

Markers of vascular stiffness (pulse wave velocity and augmentation index) were increased in donors compared to controls at 12 months but at 5 years they were not significantly different. No changes in office or ambulatory blood pressure were observed at any time point in donors or controls. At 5 years, uric acid was significantly greater in donors than controls (335 \pm 83 μ mol/L vs. 276 \pm 7, p=<0.001) and had increased after the 12 month time point despite an improvement in eGFR. At 12 months the prevalence of a detectable troponin and levels of fibroblast growth factor-23 were greater in donors compared to controls, but this effect was lost at 5 years

Conclusion: The reduction in eGFR associated with nephrectomy in living kidney donors in the absence of intrinsic renal disease or comorbidity does not lead to adverse changes in cardiovascular structure and function at 5 years. This study offers reassurance to living kidney donors and the transplant community but should prompt further work into the causes of cardiovascular disease in CKD as we have found no medium term deleterious cardiovascular effects of an isolated reduction in eGFR.