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P237 -PROGRESSION OF MYOCARADIAL FIBROSIS IN CKD

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Background— Chronic kidney disease (CKD) is a major, untreated risk factor for cardiovascular (CV) disease. Heart failure and sudden arrhythmic death account for much of the excess CV mortality in CKD. Myocardial biopsies from uraemic patients have shown cellular hypertrophy, diffuse interstitial fibrosis (DIF) and myocardial disarray. However to date it has not been possible characterise the myocardium across the spectrum of CKD in a safe way. The purpose of this study as to assess the onset and progression of myocardial fibrosis as measured by myocardial T1 mapping and extracellular volume fraction (ECV) across the spectrum of CKD.

Methods— 152 patients with CKD stage 2 to 5, without diabetes mellitus or known CV disease underwent cardiac magnetic resonance imaging (CMR 1.5T) including T1 mapping (MOLLI) and ECV. T2 mapping (T2-prepared SSFP) was used to assess myocardial free water content (MyoMaps, Siemens). Gadolinium was given if eGFR >30ml/min/1.73m2. 14 patients underwent kidney transplantation with repeat CMR at 8 weeks after transplantation to assess ECV in end-stage kidney disease. Ischaemic heart disease was excluded using either exercise stress echocardiography or 99m technetium tetrofosmin SPECT-CT. Serum biomarkers of fibrosis were also measured: P1NP, P3NP.

Results—11 patients were on haemodialysis and 3 were on peritoneal dialysis. LV volumes and LV mass, and T2 times only increased in end-stage kidney disease (ESKD). Myocardial T1 times were higher in CKD compared to controls (controls: T1 time 962±22ms), and increased progressively with worsening eGFR. ECV was higher in CKD compared to controls (controls: ECV 24±2%). Serum biomarkers of fibrosis (P1NP and P3NP) also increased with reducing eGFR. Gadolinium was administered to 68 out of 74 eligible patients with early CKD (eGFR >30ml/min): 21/68 (30%) had right ventricular insertion point (RVIP) late gadolinium enhancement (LGE); 8/68 (12%) had LGE in a non-ischaemic pattern. In the transplanted sub-cohort, 1 patient had midwall LGE and 3 had RVIP LGE.

Conclusions— For the first time, we have shown that both myocardial T1 times and ECV are elevated early in CKD, and increase progressively with declining kidney function; thereby suggesting that DIF increases steadily with decreasing GFR. This finding is supported by the observed rise in serum biomarkers of fibrosis. In ESKD, interstitial fluid as well as DIF may be contributing towards the rise in T1 times, given the observed increase in T2 times.