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The functional consequences of myocardial fibrosis in chronic kidney disease

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Background:
Advances in cardiovascular MRI (CMR) have allowed the phenotype of uraemic cardiomyopathy (UC) to be further refined with myocardial fibrosis and hypertrophy now thought to account for much of the excess cardiovascular (CV) risk imposed by CKD. The functional consequences of these imaging biomarkers have not been studied but are arguably more important in patient reported outcomes. Markers of exercise intolerance such as peak oxygen uptake (peak VO2) and % predicted peak VO2 at the ventilatory threshold (VO2VT) are associated with poor CV outcomes in renal disease. Furthermore, tissue Doppler assessment on echo which can indirectly assess left ventricular (LV) stiffness using E/e' ratio and left atrial volume are also abnormal in CKD. This study aimed to prospectively assess LV function and fibrosis on CMR with quantitative functional consequences using exercise and echocardiography.

Methods:
134 patients with CKD stage 2 to 5 pre-dialysis, without diabetes mellitus or known CV disease were studied. All patients underwent; i) CMR (1.5T) with T1 mapping (MOLLI), ii) cardiopulmonary exercise test (CPESE, GE Case ES V6.61) and (iii) Stress echocardiography with bicycle ergometer.

Results:
Myocardial T1 time had an inverse graded relationship with eGFR (Spearman correlation: r=-0.316; p<0.001). The % predicted peak VO2 and VO2VT fell with eGFR despite normal LV ejection fraction and low prevalence of LVH (9%).(Table 1) Resting myocardial strain on CMR and echocardiography were also reduced in severe CKD, while left atrial volume and BNP increased with worsening CKD. Resting E/e' increased with stage of CKD. E/e' was positively correlated with myocardial T1 time (Rest E/e': r=0.201 p=0.028; Stress E/e': r=0.235 p=0.025).

Conclusion:
Diffuse myocardial fibrosis increases with stage of CKD and is associated with reduced effort intolerance and non-invasive markers of LV stiffness. These data support the hypothesis that myocardial fibrosis is a major cause of the functional limitation in CKD.