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P194 -Endothelial Dysfunction and Blood Biomarkers in Kidney Transplant Patients

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Background:
Cardiovascular events are the commonest cause of mortality and morbidity in kidney transplant patients (KTxPs); endothelial dysfunction and consequent atherosclerosis may play a role. There is a poor understanding of how endothelial function changes with time in KTxPs and effect of novel biomarkers are unknown.

Methods: Brachial artery flow-mediated dilation (FMD) and nitroglycerin-mediated dilation (NMD) were assessed in 18 KTxPs and 17 controls at baseline and 3-6 months after. Blood biomarkers were assessed using Luminex technology multiplex assay. All subjects were recruited after written consent and all measurements were done in our vascular laboratory under standard conditions.

Results: The median time since transplantation was 86 months (interquartile range 123 months). There were more dyslipidaemics in KTxPs compared to controls (10 vs. 3; P=0.02), and eGFR was lower (67.61 ± 20.25 vs. 97.59 ± 15.59; P<0.01). There was no difference in age (51.28 ± 13.29 vs. 45.82 ± 10.85; P=0.19), body mass index (25.56 ± 5.18 vs. 24.59 ± 2.59; P=0.49), diabetes status (3 vs. 0; P=0.08), systolic blood pressure (131.94 ± 11.79 vs. 125.53 ± 12.39 mmHg; P=0.13), diastolic blood pressure (82.17 ± 9.22 vs. 77.24 ± 7.61 mmHg; P=0.10) and vitamin D (57.56 ± 25.21 vs. 43.65 ± 22.03 nmol/L; P=0.09).

Fibroblast growth factor 23 (FGF-23) was higher (145.91 ± 176.79 vs. 35.78 ± 58.32 pg/ml; P=0.02) in KTxPs, matrix metalloproteinase 2 (MMP-2) was numerically higher (744.38 ± 578.26 vs. 552.93 ± 363.32 pg/ml; P=0.25), but statistically not significant. Baseline FGF-23 correlated with MMP-2 (r = 0.52; P=0.03).

No significant difference existed in vascular markers between KTxPs and controls at baseline: FMD (4.34 ± 3.45 vs. 4.63 ± 3.02 %; P=0.79), NMD (15.15 ± 6.08 vs. 16.00 ± 5.47 %; P=0.67). Markers did not change in controls upon follow-up. In KTxPs, FMD decreased (-1.52 ± 2.74 %; P=0.03).

Conclusions: Endothelial dysfunction worsened in stable KTxPs upon long-term follow-up. FGF-23 and MMP-2 may contribute to elevated cardiovascular risk by adversely affecting endothelial function in KTxPs.