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CHRONIC KIDNEY DISEASE. PATHOPHYSIOLOGY, PROGRESSION & RISK FACTORS - 1

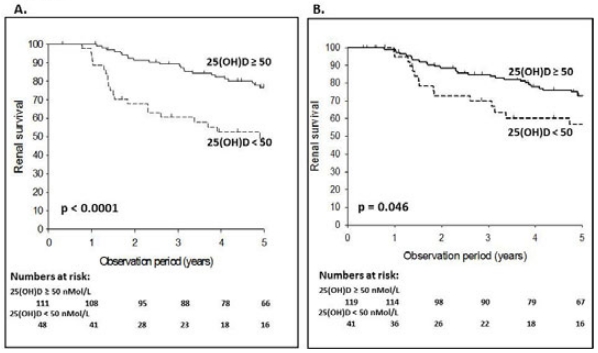
FP282 **NORMAL 25-HYDROXYVITAMIN D LEVELS ARE ASSOCIATED WITH LESS PROTEINURIA AND ATTENUATE RENAL FAILURE PROGRESSION IN CHILDREN WITH CHRONIC KIDNEY DISEASE**

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Introduction and Aims: Angiotensin converting enzyme inhibitors (ACEi) for renin-angiotensin-aldosterone system blockade are routinely used to slow CKD progression. Vitamin D may promote renoprotection by suppressing renin transcription through cross-talk between RAAS and vitamin D - Fibroblast Growth Factor-23 - klotho pathways. We hypothesise that vitamin D levels influence proteinuria and CKD progression in children.
Methods: We performed a post-hoc analysis of the ESCAPE (Effect of Strict Blood Pressure Control and ACE Inhibition on Progression of CKD in Pediatric Patients) cohort. Serum 25-hydroxyvitamin D (25(OH)D) and klotho were measured at baseline and after a median of 8 months on ACEi.
Results: Of the 167 children (median age 11.4 years, 96 males), 129 had congenital anomalies of the kidneys and urinary tract, 15 glomerulopathy and 23 other non-proteinuric renal diseases. Median urinary protein/creatinine ratio was 0.78 (IQR 0.23 - 2.0) and none had nephrotic range proteinuria.Children with lower 25(OH)D levels had higher urinary protein/creatinine ratios both at baseline (p=0.03) and follow-up (p=0.02). Serum 25(OH)D did not correlate with serum albumin levels, implying that the association between 25(OH)D and proteinuria is not due to urinary loss of vitamin D binding protein. 25(OH)D levels ≤50nMol/L associated with higher diastolic BP (p=0.004). The annualized loss of eGFR was inversely associated with baseline 25(OH)D (p=0.0003, r=0.32). 5-year renal survival was 75% in patients with baseline 25(OH)D ≥50 nMol/L compared to 50% in those with lower 25(OH)D (p<0.0001); Figure 1A. This renoprotective effect remained significant, but attenuated, on ACEi therapy (p=0.05); Figure 1B.

Figure 1



Renal survival increased by 7.9% for each 10 nMol/L increase in 25(OH)D (p=0.034), independent of eGFR, proteinuria and underlying renal diagnosis. ACEi treatment significantly increased FGF-23 and klotho (p<0.001 for both), without notable changes in serum calcium or phosphorus.
Conclusions: In children with CKD 25(OH)D levels ≥50nMol/L were associated with reduced proteinuria and greater preservation of renal function, even in the presence of ACEi treatment. Prospective studies on the renoprotective effects of vitamin D are required.

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