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PRESENTATION TYPE: Oral Presentation

CURRENT CATEGORY: Renal/Electrolyte and Hypertension

TITLE: CIRCULATING 24,25(OH)2 D CONCENTRATIONS DURING TREATMENT OF VITA DEFICIENCY.

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ABSTRACT BODY:
Purpose of Study: We have previously reported resistance to correction of vitamin D deficier cholecalciferol therapy in CKD patients, as compared to subjects with normal kidney function 2012). Elevated FGF23 levels in CKD stimulate Cyp24a1 expression, suggesting that Cyp24- catabolism of 25(OH)D may contribute to this resistance. To test this possibility 24,25(OH)D2 measured before and after treatment of vitamin D deficiency with cholecalciferol.

Methods Used: Prospective study of 28 patients with 25OHD level < 20 ng/ml who received IU/week of cholecalciferol for 8 weeks. CKD (n=14) were matched with non-CKD group (n=14 ,race, and diabetes. Response to cholecalciferol was assessed by measuring the changes in 25(OH)D and 24,25(OH)2D3 levels.

Summary of Results: There were no significant differences between CKD and non-CKD pat baseline concentrations of serum albumin, 25(OH)D,1,25(OH)2D , 24,25(OH)2D3. Baseline ir (PTH) and FGF-23 levels were higher in CKD patients. In a multivariable analysis low pre-trea 24,25(OH)2D3 level was predicted by low 25(OH)D, high PTH and high FGF23 levels. Cholec treatment resulted in increases in serum 25(OH)D (18.6 ± 8 ng/ml vs 12.2 ± 9, p<0.03), 1,25((32.3 pg/ml vs 4.3 ± 23.6, p=0.31), and 24,25(OH)2D3 (1.14 ± 0.89 ng/ml vs 1.02 ± 0.74, p=0.7 CKD and CKD, respectively. 24,25(OH)D2 levels were not higher in CKD, either before or afte cholecalciferol, despite higher FGF23. PTH levels decreased after treatment in CKD patients pg/ml, p<0.05), but not in non-CKD patients (-10±25, p=0.16). Cholecalciferol also resulted in in serum FGF23 (81 ± 35 pg/ml vs 125± 42, p=0.052) in non-CKD, but was unchanged in CKI multivariable analysis, low post-treatment serum 24,25(OH)2D3 level was predicted by low ba 24,25(OH)2D3 (p= 0.09), low post-treatment 25(OH)D (p<0.03), and high baseline PTH (p<0.

Conclusions: 24,25(OH)2D3 increases with cholecalciferol therapy in both CKD and non-CK consistent with substrate dependent production. Despite decreased nephron mass in CKD, th similar increment in 24,25(OH)2D3 following cholecalciferol.

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