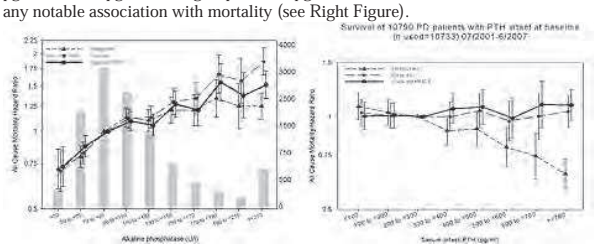
SA-PO2150

Association of Mortality with Serum Alkaline Phosphatase (AlkPhos) and Parathyroid Hormone (PTH) in Chronic Peritoneal Dialysis (CPD) Patients Kamyar Kalantar-Zadeh,[1] Uyen Duong,[1] Csaba P. Kovesdy,[3] Allen R. Nissenson,[4] Keith C. Norris,[5] Rajnish Mehrotra.[2] *[1]Harold Simmons Center, Harbor-UCLA Medical Center, Torrance, CA; [2]David Geffen School of Medicine at UCLA, Los Angeles, CA; [3]Salem VA Medical Center, Salem, VA; [4]Davita, Lakewood, CO; [5]King-Drew School of Medicine, Lynwood, CA.*

Background: Serum AlkPhos may be a better mortality predictor than PTH in dialysis patients (pts). We examined this hypothesis in CPD pts, since the spectrum of bone disease may vary by dialysis modality. **Methods:** We examined cohort of all CPD pts from July 2001 through June 2007. **Results:** We identified 12,422 CPD pts who had baseline AlkPhos and PTH measures. They were 54±16 years old, 47% women and 23% African Americans. Serum AlkPhos was divided into 10 preselected groups by increments of 20 U/L. Higher AlkPhos was associated with increased death risk including adjustment for case-mix and malnutrition-inflammation complex syndrome. Death hazard ratios (HR) and 95% CIs for AlkPhos groups >130 U/L were 1.3(1.1-1.4), 1.2(1.1-1.4), 1.6(1.3-1.8), 1.4(1.1-1.7), and 1.5(1.3-1.7) respectively. We also found that lower AlkPhos levels were independently associated with better survival: Death HRs (95% CI) for AlkPhos in 50-<70 and ≤50 U/L were 0.7(0.6-0.9), 0.8(0.7-0.9), respectively (see Left Figure). By contrast, eight a preselected groups of PTH <100 pg/ml to ≥ 700pg/ml and 6 groups of 100 pg/ml increments in-between did not show any notable association with mortality (see Right Figure).



Conclusions: In this CPD cohort, increase in AlkPhos (≥130U/L) was associated with increase in all-cause mortality and decrease in Alkphos (≤70U/L) with greater survival, whereas PTH did not exhibit death association. Additional studies to examine utility of AlkPhos for clinical decision making in CPD patients are indicated.

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SA-PO2151

Evaluation of 16 Months of Clinical Use of Cinacalcet in Haemodialysis and Peritoneal Dialysis Patients: An Observational Study in Belgium (ECHO-B) Frederic D. Debelle,[1] Gert Meeus,[2] Michel Dhaene,[1] Georges F. M. Cornet,[3] Xavier Warling,[4] Sofie Jamar,[5] Evemie Schutyser,[6] Michel Y. Jadoul.[7] *[1]RHMS, Baudour; [2]AZ Groeninge, Kortrijk; [3]CH Peltzer-La Tourelle, Verviers; [4]CHR de la Citadelle, Liège; [5]Imelda, Bonheiden; [6]Amgen Belgium; [7]UCL St-Luc, Brussels.*

In this Belgian observational study we investigated the effectiveness and treatment patterns of cinacalcet in dialysis patients with secondary hyperparathyroidism (SHPT). Data from 81 SHPT patients from 20 centers were collected from 6 months before to 16 months after cinacalcet initiation. Results are presented as means±SD, unless indicated otherwise. Follow-up time after cinacalcet initiation was 15.4±2.7 months. Patients were 48% male, aged 58±16 (SD) years and on dialysis for 5.2±5.7 years. They had markedly elevated intact parathyroid hormone (iPTH) levels (860±526 pg/ml) and elevated phosphorus (P) levels (5.7±1.5 mg/dL) whereas albumin-corrected calcium (Ca) concentration was 9.4±0.9 mg/dL at baseline (cinacalcet initiation).

At baseline, the proportion achieving the NKF-K/DOQI™ targets was: PTH (1%), P (40%), Ca (54%), and CaxP (48%). These values were 25%, 47%, 48% & 81% at 6 months, and 42%, 52%, 34% & 80% at 16 months after cinacalcet initiation, respectively. The proportion of patients reaching both the iPTH and CaxP targets was 21% at 6 months and 33% at 16 months *versus* 2% at baseline. Maximum reductions of Ca (-7.5%), P (-11.9%) and CaxP (-17.6%) were seen at 4 months, whereas iPTH levels were even further reduced by 36% at 4 months and 51% at 16 months. The primary endpoint (achievement of an iPTH level between 150 and 300 pg/ml and/or a reduction of 30% or more within 4 months of cinacalcet start) was achieved in 80% of patients.

Mean and median Cinacalcet doses were 48 and 30 mg in the 6th month, and 52 and 51 mg in the 16th month, respectively. Sevelamer use decreased upon cinacalcet initiation, while doses of Ca-containing P binders slightly increased. The data from this observational study are in line with published randomized controlled trials and larger observational studies, and further support the use of cinacalcet for the treatment of SHPT in dialysis patients.

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SA-PO2152

Effect of Intravenous Iron Therapy on Serum FGF23 Levels and Mineral Metabolism in ESRD Patient Undergoing Hemodialysis Yoko Takeda,[1] Hirotaka Komaba,[2] Shunsuke Goto,[1] Keiji Kono,[1] Kentaro Nakai,[1] Hideki Fujii,[1] Shinichi Nishi,[1] Masafumi Fukagawa.[2] *[1]Division of Nephrology and Kidney Center, Kobe University Graduate School of Medicine, Kobe, Hyogo, Japan; [2]Division of Nephrology, Endocrinology and Metabolism, Tokai University School of Medicine, Isehara, Kanagawa, Japan.*

INTRODUCTION AND AIMS: Fibroblast growth factor 23 (FGF23) induces urinary phosphate excretion, suppresses 1,25-dihydroxyvitamin D synthesis, and inhibits parathyroid hormone (PTH) secretion. Recent data suggest that FGF23 plays a role in the development of iron-induced hypophosphatemia in subjects with normal kidney function. The aim of this study was to examine the effect of intravenous iron therapy on serum FGF23 levels and mineral metabolism in patients undergoing hemodialysis.

METHODS: This prospective study enrolled 27 patients who were receiving hemodialysis for more than three months and had iron-deficiency anemia defined by a hemoglobin concentration less than 10.5 g/dl and serum ferritin less than 100 ng/ml. Intravenous saccharated ferric oxide(SFO) at a dose of 40 mg was administered thrice weekly over three weeks. Serum FGF23, intact PTH and other parameters were prospectively monitored for five weeks.

RESULTS: Intravenous iron therapy resulted in a significant increase in hemoglobin and ferritin levels at wk 3 (10.0 ± 0.5 g/dl to 10.6 ± 0.5 g/dl; P < 0.001, 30.1 ± 22.7 to 153.83 ± 83 ng/ml; P <0.001, respectively). Serum FGF23 increased from 4906 ± 6201 pg/ml at baseline to 8824 ± 11041 pg/ml at wk 3 (P = 0.031), whereas intact PTH decreased from 146 ± 109 pg/ml to 109 ± 77 pg/ml (P <0.001). TRACP-5b decreased from 477 ± 295 mU/dL to 412 ± 254 mU/dL (P <0.001) at wk 3. These levels gradually returned to baseline by wk 5. There were no significant changes in serum calcium and phosphorus during the study period.

CONCLUSIONS: Intravenous iron therapy results in further increase in FGF23 levels in hemodialysis patients. This increase does not induce hypophosphatemia in the absence of functioning kidney, but may result in transient PTH suppression and its related decreased bone resorption.

Disclosure of Financial Relationships: nothing to disclose

SA-PO2153

Racial Differences in Vitamin D, Parathyroid Hormone and Fibroblast Growth Factor-23 Levels in Patients with Severe Chronic Kidney Disease Anna Jeanette Jovanovich,[1] Michel B. Chonchol,[1] Alfred K. Cheung,[2,4] James S. Kaufman,[3] Tom H. Greene,[4] William L. Roberts,[4] Gerard John Smits,[1] Jessica B. Kendrick.[1] *[1]University of Colorado Denver Health Sciences Center, Denver, CO; [2]VASLCHCS, Salt Lake City, UT; [3]VA Boston Healthcare System, Boston, MA; [4]University of Utah, Salt Lake City, UT.*

Purpose: Abnormalities of mineral metabolism have not been described across races in patients with severe CKD.

Methods: Study was conducted among patients with severe CKD, but not on dialysis, who participated in the Homocysteine in Kidney and End Stage Renal Disease study. 25-hydroxyvitamin D (25(OH)D), calcitriol (1,25(OH)2D), intact parathyroid hormone (iPTH), and fibroblast growth factor (FGF-23) levels were measured in plasma samples. Multivariable regression analyses were performed to examine the association between race and vitamin D, iPTH, and FGF-23 levels.

Results: There were a total of 1099 patients with an eGFR of 18 ± 6.5 mL/min/1.73m2. 57% of the cohort was non-Hispanic white (NHW), 26% was non-Hispanic black (NHB) and 17% were categorized as other races. NHB had the lowest 25(OH) D levels when compared to NHW and others (15.8±9.0 vs. 22±10 vs. 24±10 ng/mL respectively; p<0.0001). However, there was no significant difference in 1,25(OH)2D levels among races. NHB had higher iPTH levels than NHW and others (248±208 vs. 167±131 vs. 210±156 pg/mL respectively; p<0.0001). The median [IQR] FGF-23 levels among NHB, NHW and others were 324 [181-655], 431 [232-1026], and 364 [219-895] RU/mL, respectively (p=0.0001). After adjustment for demographics, cardiovascular risk factors and eGFR, NHB was independently associated with lower 25(OH)D (β= -0.25; p<0.0001) and higher iPTH (β=0.22; p<0.0001) levels than non-Blacks. NHB was independently associated with increased iPTH when further adjusted for calcium, phosphorus, 25(OH)D, 1,25(OH)2D and FGF-23 suggesting that other mechanism play a role in elevated iPTH levels in NHB. There were no racial differences in multivariable-adjusted 1,25(OH)2D and FGF-23 levels.

Conclusions: Low 25(OH)D and elevated iPTH levels are more severe in NHB when compared to non-blacks with severe CKD.

Disclosure of Financial Relationships: nothing to disclose

SA-PO2154

Preliminary Validation of Three Commercially Available Assays for Measurement of Human Plasma FGF23 Sandra M. Malakauskas,[1] Jun Ling Lu,[2] Ali Iranmanesh,[1,3] Csaba P. Kovesdy.[1,3] *[1]Salem VA Medical Center, Salem, VA; [2]Salem Research Institute, Salem, VA; [3]University of Virginia, Charlottesville, VA.*

FGF23 is a bone-derived peptide hormone that is emerging as an integral regulator of phosphorus balance and vitamin D hydroxylation, yet its clinical significance in CKD and ESRD is poorly understood. The purpose of this study is to perform preliminary validation of 3 commercially available assays for measurement of FGF23 in human plasma