

patients who are on hemodialysis. But higher cholesterol level does not seem to effect on raising mortality or cardiovascular morbidity and CAPD failure. On the contrary, lower serum cholesterol level in CAPD patients tends to raise mortality and morbidity due to poor nutritional status.

Methods: This study is a retrospective study designed to evaluate the effect of cholesterol level, statin on CAPD outcome and mortality. Patients who were on peritoneal dialysis for at least 6 months since March 1st, 2000 were included. A total of 467 patients were enrolled in this study. Patients' biological parameter, biochemical parameter and morbidity/mortality during CAPD maintenance period were collected.

Results: Patients whose initial cholesterol level were above 240 mg/dL shows significantly low CAPD failure rate compared to patients whose initial cholesterol level were below 200 mg/dL (OR=0.469, $p=0.049$). Patients whose average LDL-cholesterol during CAPD period were over 100mg/dL showed significantly higher mortality compared to patients whose initial LDL-cholesterol level were below 100mg/dL (OR=1.848, $p=0.024$). Patients whose compliance to statin during CAPD period was over 80% showed significantly low mortality compared to patients who did not take statin during CAPD period (OR=0.556, $p=0.020$). Patients showed no significant difference in mortality due to total cholesterol, HDL cholesterol levels and patients showed no significant difference in CAPD failure due to HDL/LDL cholesterol, statin usage.

Conclusions: Usually compared to HD or CKD patients, serum total cholesterol level in CAPD patients was lower significantly. However, triglyceride level in CAPD patients was significantly higher than CKD or HD patient. In this study, the use of statins unrelated to cholesterol level was significantly effective to clinical outcomes in CAPD patients. To verify this results the larger size and prospective study is needed.*Corresponding author.

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BODY COMPOSITION IN PREDIALYSIS PATIENTS ON A LOW PROTEIN DIET SUPPLEMENTED WITH KETOANALOGUES OF ESSENTIAL AMINOACIDS VERSUS A FREE DIET

Shutov Evgeny, Fedorov Dmitry

Department of Nephrology, Clinical Hospital named after S.P. Botkin, Moscow, Russia

More than a half of patients starting dialysis are found to have a poor predialysis nutritional status. A low-protein diet (LPD) delays CKD progression but occasionally lead to some changes in body composition and malnutrition.

We performed a prospective, open-label, parallel, randomized and controlled trial to compare a body composition and biochemical parameters of CKD patients who were on LPD supplemented with ketoanalogues of essential aminoacids (KA) with those on a free diet (FD). Biochemical and bioimpedance parameters were analyzed in CKD patients every month before initiation of dialysis. Impedance measurements were performed with a multifrequency bioelectrical impedance analyzer. Group I ($n=14$ patients, 8 male, 6 female; 53 ± 12 y.o.; glomerular filtration rate (GFR) $18,8 \pm 6,3$ ml/min (MDRD formula)) having LPD (0.5 g/kg/day protein) supplemented with KA (1 tablet/5 kg of body weight a day) was compared with matched Group II on FD ($n=15$ CKD patients with GFR $17,8 \pm 5,9$ ml/min). Protein intake was lower in patients on LPD ($0,5 \pm 0,1$ g/kg/day protein) than on FD ($1,14 \pm 0,21$ g/kg/day; $p < 0.0001$). The declining slopes of GFR during LPD + KA period were significantly lower than those during FD, so Group I was invited to dialysis later - after $32,4 \pm 12,7$ months in contrast with Group II in $14,5 \pm 7,7$ months ($p < 0.0001$). Albumin and pre-albumin levels remained stable in Group I, but they decreased in Group II ($\Delta = -0,93 \pm 0,21$ g/l, $\Delta = -0,15 \pm 0,12$ g/l respectively; $p < 0.001$) at the start of dialysis. The body composition didn't change significantly in patients on LPD while in patients on FD their lean body mass decreased by $3,5 \pm 2,3$ kg ($p < 0,001$), body fat mass declined by $1,5 \pm 0,4$ kg ($p < 0,01$), simultaneously extracellular water increased $+1,5 \pm 0,9$ l, $p < 0,05$.

In conclusion, LPD and KA delayed CKD progression without nutritional status and body composition deterioration in contrast to FD. Controlled LPD with KA should be widely recommended as a safer dietary method for pre-dialysis CKD patients than FD.

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EVALUATION OF SERUM PHOSPHORUS LEVELS AND OBESITY

John Sim¹, Ning Smith¹, Joanie Chung¹, Kamyar Kalantar-Zadeh²

¹Research & Evaluations Kaiser Permanente SoCal, Pasadena, CA, USA

²Harbor UCLA Medical Center, Torrance, CA, USA

Higher dairy intake overall has been associated with lower risk for the metabolic syndrome while higher phosphorus (phos) intake has conversely shown greater risk for obesity and the metabolic syndrome. The higher phos content in food preservatives and food flavorings and the greater phos absorption from consumption of animal products overall contribute to the poor dietary habits linked with the rising rate of obesity in the USA. We sought to determine whether higher levels of serum phos are associated with obesity within a large ethnically diverse population of patients who are free of kidney disease.

In the period 1/1/2007-12/31/2011, all subjects age > 17yrs with measured serum phos, documented eGFR ≥ 60 ml/min, and documented body mass index (BMI) were identified from the Kaiser Permanente So Cal healthplan. Subjects were categorized into population based quartiles from phos values. Obesity was defined as BMI > 25. Race/ethnicity, HTN, and DM were extracted from electronic medical records and the use of ICD codes.

A total of 88,094 subjects were analyzed. In the univariate regressions, higher phos quartiles compared to the lowest quartile, had OR (95% CI) for obesity of 1.09(1.06-1.12), 1.06(1.03-1.08), and 1.05(1.02-1.07) respectively for phos quartiles 3.1-3.4(mg/dl), 3.5-3.9, 4.0-5.7 vs 1.9-3.0. Linear regressions showed every 0.5mg/dl phos increase demonstrated OR of 1.02 (1.01-1.02) for obesity. These differences were not sustained in the multivariable analyses. However, Blacks (1.29) and Hispanics (1.48) had higher risk for obesity while risk for Asians (0.38) were lower compared to whites ($p < 0.05$). Presence of DM (2.00) and HTN (2.16) also had higher OR for obesity. In subjects without kidney disease, serum phos levels may be reflective of poor dietary habits and may represent a risk factor for the metabolic syndrome.

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USEFULNESS OF NUTRITIONAL PARAMETERS BASED ON CREATININE KINETIC MODEL IN PREDICTING PROGNOSIS IN SEVERE AKI PATIENTS REQUIRING CRRT

Joan Ho Song, Seong Bin Hong, Seoung Woo Lee

Dept. of Int. Med, Inha University School of Medicine, Incheon, Korea

Malnutrition and protein catabolism have been known as important prognostic factors in AKI patient. The purpose of the present study was to evaluate the usefulness of application of creatinine (CKM) and urea kinetic modeling (UKM) based on the mass balance equation during CRRT treatment and their prognostic significance in severe AKI patients.

Urea generation rate (UGR; mg/min) and Cr production rate (Pc; mg/day) were calculated by measuring urea and Cr production/removal balance from patients' serum, the spent dialysate from CRRT and, if any, urine in 50 ICU patients with severe AKI receiving CRRT. Protein catabolic rate (nPCR; g/kg/d) and lean body mass (LBM; kg) were calculated according to the equations; PCR = [(9.35 x UGR)/BW] + 0.17, LBM = (0.029xPc) + 7.38. Pc was standardized with body weight and converted into Cr Index (CI; mg/kg/d).

The impact of these CKM and UKM parameters on the prognosis in addition to conventional subjective global assessment (SGA) and other known traditional prognostic factors was analyzed. Overall survival of the subjects was 35.3%. Non-survivors showed not only significantly higher APACHE II and chronic comorbidities scores but also severely-malnourished state demonstrated by lower serum albumin, BMI, CI, and LBM. It was revealed that independent parameters associated with mortality were poor SGA (C; Hazard ratio 3.5), low CI (< 11 mg/kg/d; HR 1.8), and high comorbidities score (≥ 3 ; HR 1.9).

In conclusion, nutritional state and chronic comorbidities were major factors predicting the clinical outcome of severe AKI patients requiring CRRT. CKM was a simple and useful method in the assessment of nutritional state during CRRT treatment. Low creatinine production reflecting poor nutrition and protein reserve was associated with poor prognosis in severely ill ARF patients.

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EFFECT OF ANTIOXIDANT TO PROGRESSION OF DIABETIC NEPHROPATHY IN DIABETIC RAT

Ki Sung Kang¹, Noriko Yamabe², Woojung Lee¹, Seungyong Lee¹, Sunam Kim¹, Dae-Woon Eom³, Kyung Il Song^{4,*}