NUTRITION, INFLAMMATION AND OXIDATIVE STRESS - CKD 1-5

SAP261  OXIDATIVE STRESS AND SYSTEMIC INFLAMMATION AS IMPORTANT COMPONENTS OF CARDIAC VALVE CALCIFICATION IN PREDIALYSIS PATIENTS

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1Ternopil University Hospital, Ternopil, Ukraine

Introduction and Aims: According to the modern tendencies, the important formation factors of cardiovascular calcification in chronic kidney disease (CKD) are oxidative stress (OS) and chronic inflammation. However, the role of the indicated processes in mechanisms of cardiac valve calcification (CVC) in patients under the predialysis period of CKD were not asserted enough. In this conditions, it is also reasonable to research the functional activity of endothelium, especially of nitric oxide (NO) system, since endothelial dysfunction is one of the key mechanisms, which mediated inflammatory effects on the cardiac valve apparatus. The purpose of the current study was (1) to determine the role of activation of free radical oxidation of lipids and systemic inflammation in mechanisms of valve calcification in predialysis CKD patients, and (2) to establish in CVC group the relationship between the inflammatory markers and NO system state.

Methods: We enrolled 167 (male/female, 78/89; age, 48.7 ± 13.2 years; eGFR-MDRD, 51.0 ± 28.2 ml/min per 1.73 m²) predialysis (stage I: 8.4%, stage II: 28.1%, stage III: 38.9%, stage IV: 18.0%, stage V: 6.6%) CKD patients. All subjects underwent echocardiographical examination for detection of valve calcification. Plasma content of malondialdehyde (MDA), superoxide dismutase (SOD) and catalase (CAT) activities, ceruloplasmin (CP) and glutathione (GSH) concentrations as indices of pro/antioxidant system were determined using the standard methods. Inflammatory markers were performed by evaluating the serum levels of C-reactive protein (CRP) (imunoturbidimetric method), fibrinogen (gravimetric analysis) and circulating immune complexes (CICs) (polyethylene glycol precipitation test). Plasma content of nitrates (NO2−) (Green L.C. et al., 1982) was measured as marker of NO system activity. Data are presented as mean ± SD. Used nonparametric statistics methods: Mann-Whitney U-test in order to compare indices in two groups, Spearman’s rank R correlations for establishment of presence and strength of the relationship between the research parameters.

Results: CVC was detected in 28.7% of predialysis CKD patients: isolated calcification of mitral valve – in 6.0%, aortal valve – in 7.2%, both valves – in 15.6%. In CVC group indices of MDA (p < 0.001), CP (p < 0.001), CRP (p < 0.001), fibrinogen (p = 0.005), CICs (p = 0.010) were higher, and SOD (p < 0.001), CAT (p = 0.061), GSH (p = 0.005) – lower compared to the group without calcification. Predialysis CKD patients with valve calcification had lower content of NO2−: (0.053 ± 0.016 vs. 0.070 ± 0.016 mmol/L, Z = 4.999, p < 0.001) than subjects without calcification. For the first time it was established that in CVC group (n = p;48) NO2− level was correlated with CRP (R = -0.565, p < 0.001), fibrinogen (R = -0.415, p = 0.003) and CICs (R = -0.512, p < 0.001) concentrations.

Conclusions: (1) CVC in predialysis CKD patients is combined with the development of OS and systemic inflammation. (2) Valve calcification under the predialysis period of CKD is characterized by the reduced content of stable metabolite of NO – NO2−, which in turn is closely related with the inflammatory parameters. (3) Availability of complex of metabolic disturbances in predialysis patients with CVC points towards the necessity of the search of means with normalizing influence on the level of lipid peroxidation, antioxidant defense, inflammatory processes and NO system state for prevention and treatment of the indicated cardiac damages.

SAP262  GERIATRIC NUTRITIONAL RISK INDEX MAY BE A SIGNIFICANT PREDICTOR OF MORTALITY IN KOREAN HEMODIALYSIS PATIENTS: A SINGLE CENTER STUDY

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1Kosin University College of Medicine

Introduction and Aims: The GNRI is a very simple and objective method to assess nutritional condition, utilizing only three objective parameters: body weight, height and serum albumin values. To date, there have been few longitudinal studies that have used GNRI to predict mortality in chronic hemodialysis patients. In the present study, we validated whether using GNRI as a nutritional screening tool could be a clinical predictor of mortality in Korean hemodialysis patients.

Methods: We examined the GNRI of 120 maintenance hemodialysis patients and followed these patients for 120 months. Predictors for all-cause death were examined using life table analysis and the Cox proportional hazards model

Results: Life table analysis revealed that subjects with a GNRI < 90 (n = 19) had a marginally lower survival rate than did those with a GNRI = 90 (n = 101) (Wilcoxon test, P = 0.048). Multivariate Cox proportional hazards analyses demonstrated that the GNRI was a significant predictor of mortality [hazard ratio (HR) 0.966, 95% confidence interval (CI) 0.945–0.995, P = 0.018], after adjusting for age, sex, presence of type 2 diabetes mellitus, and body weight.

Conclusions: These results demonstrate that the GNRI may be a significant predictor of mortality in Korean hemodialysis patients.

SAP262 Table 1 Clinical characteristics of 120 chronic hemodialysis patients according to GNRI

<table>
<thead>
<tr>
<th>Variable</th>
<th>GNRI &lt; 90</th>
<th>GNRI ≥ 90</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>56.7 ± 15.9</td>
<td>56.1 ± 12.1</td>
<td>0.643</td>
</tr>
<tr>
<td>Male/Female</td>
<td>9/10</td>
<td>10/9</td>
<td>0.640</td>
</tr>
<tr>
<td>Diabetes (%)</td>
<td>127</td>
<td>50/51</td>
<td>0.275</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>51.3 ± 11.6</td>
<td>60.3 ± 12.5</td>
<td>0.016</td>
</tr>
<tr>
<td>Body mass index</td>
<td>18.6 ± 0.6</td>
<td>22.3 ± 0.5</td>
<td>0.016</td>
</tr>
<tr>
<td>Albumin (g/dL)</td>
<td>3.1 ± 0.4</td>
<td>3.3 ± 0.8</td>
<td>0.005</td>
</tr>
<tr>
<td>CRP (mg/dL)</td>
<td>1.4 ± 0.5</td>
<td>1.2 ± 0.4</td>
<td>0.005</td>
</tr>
<tr>
<td>SOD (u/L)</td>
<td>27.9 ± 8.2</td>
<td>32.6 ± 7.8</td>
<td>0.001</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>149 ± 29</td>
<td>141 ± 25</td>
<td>0.018</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>86 ± 11</td>
<td>87 ± 11</td>
<td>0.721</td>
</tr>
</tbody>
</table>

SAP262 Table 2. Multivariate Cox proportional hazards analysis of mortality

<table>
<thead>
<tr>
<th>Variable</th>
<th>Hazard ratio (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1.012 (0.994–1.030)</td>
<td>0.183</td>
</tr>
<tr>
<td>Sex</td>
<td>1.131 (0.710–1.800)</td>
<td>0.605</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1.546 (0.934–2.106)</td>
<td>0.167</td>
</tr>
<tr>
<td>CRP</td>
<td>0.969 (0.945–0.995)</td>
<td>0.018</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>1.035 (1.010–1.067)</td>
<td>0.002</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>0.914 (0.721–1.158)</td>
<td>0.456</td>
</tr>
<tr>
<td>nPCR</td>
<td>1.187 (0.831–1.367)</td>
<td>0.283</td>
</tr>
<tr>
<td>Phosphate</td>
<td>1.800 (0.460–3.619)</td>
<td>0.329</td>
</tr>
<tr>
<td>SBP</td>
<td>1.005 (0.976–1.034)</td>
<td>0.755</td>
</tr>
</tbody>
</table>

PCR : normalized protein catabolic rate
SBP : systolic blood pressure

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Introduction and Aims: Cardiovascular events are the main cause of death in patients with chronic kidney disease (CKD). Lipoproteins play an important role in the regulation of vascular integrity. Recent evidence suggests that urea-driven carbamylation of lysine residues may affect the functional properties of lipoproteins, however the effect on endothelial function is unknown. We therefore examined the effect of carbamylated low density lipoprotein (cLDL) on endothelial function.

Methods: cLDL from healthy donors was isolated by sequential ultracentrifugation. cLDL was carbamylated ex vivo using potassium cyanate. The degree of carbamylation and oxidation was assessed by ESI-MS/MS and Oxidized LDL assay, respectively. Vascular reactivity after treatment with native (nLDL) or carbamylated (cLDL) LDL was examined in organ chamber experiments using aortic rings of wildtype or lox-1 transgenic mice. Superoxide and nitric oxide production in aortic rings and human aortic endothelial cells (HAEC) was determined using electron spin resonance (ESR) spectroscopy. Activation of endothelial NO synthase (eNOS) was assessed by western blot techniques. In HAEC, silencing of lox-1 was performed using lox-1 specific siRNA.

Results: Carbamylation of LDL resulted in carbamyl-lysine levels comparable to those in patients with CKD without relevant oxidation. cLDL impaired endothelial-dependent relaxation of aortic rings, whereas nLDL had no effect. Addition of superoxide dismutase/catalase could restore vascular relaxation after cLDL treatment, indicating an important role of superoxide production in cLDL-mediated endothelial dysfunction. Indeed, cLDL directly induced superoxide production in aortic rings and HAEC by uncoupled eNOS. Interestingly, cLDL-mediated endothelial dysfunction was enhanced in LOX-1 transgenic mice, revealing LOX-1 as receptor for cLDL. Accordingly, siRNA-mediated knockdown of LOX-1 in HAEC could improve endothelial NO production and attenuate superoxide release.

Conclusions: Our data demonstrate for the first time that carbamylated LDL, as it is produced in patients with CKD, induces endothelial dysfunction. The effect of carbamylated LDL may contribute to the high cardiovascular burden of these patients.
by Weir and should be a target in the nutritional treatment of this population, aiming CVD prevention.

**SAP266 ESTIMATION OF TOTAL ADIPOSE TISSUE IN HEMODIALYSIS PATIENTS USING BIOIMPEDANCE TECHNIQUES**

Fansan Zhu1, George Kayser2, Peter Kotanko1, Samer R. Abbas1, Yanna Dou1, Steven Heymsfield6 and Nathan W Levin3

1Renal Research Institute, 2University of California Davis, Davis Ca USA, 3Renal Research Institute, New York USA, 4Fans of the average sum of BM, 5Renal Research Institute, Beth Israel Medical Center, New York, USA, 6St. Lukes Hospital, New York, USA

**Introduction and Aims:** Assessment of total adipose tissue (TAT) and fat free mass (FFM) plays an important role in predicting morbidity and mortality in hemodialysis (HD) patients. Traditionally, fat mass is estimated by differences between body mass (BM) and FFM assuming a constant (0.73) hydration of FFM. However, variability of fluid overload in HD patients may affect the degree hydration of FFM. The aim of this study was: 1) to identify factors affecting this ratio of total body water (TBW) to FFM and 2) to compare TAT determined by bioimpedance and magnetic resonance imaging (TATMRI).

**Methods:** Forty four HD patients (age 54.4 ± 10.6 years; 26 male; body mass 78.5 ± 17 kg) were studied with MRI and bioimpedance pre HD. Whole body bioimpedance spectroscopy (wBIS, Hydra 4200) was used to obtain whole body extracellular (wRe) and intracellular (wRi) resistance; calf normalized resistivity (CNR) was determined by calf bioimpedance. Whole body extracellular (wECV) and intracellular (wICV) volume were calculated and wTBW calculated as the sum of wECV and wICV. TATMRI was defined as subcutaneous adipose tissue (SAT) plus visceral adipose tissue (VAT) measured by MRI. Since adipose free mass (AFM) should approximate FFM, we set FFM = BM- TATMRI and the ratio of wTBW to FFM assuming a constant (0.73) hydration of FFM. However, variability of fluid overload in HD patients may affect the degree hydration of FFM. The best correlation coefficient and smallest SD of bias was obtained using the regression model (eTAT4) based on body mass, age, BMI, wRe and wRi variables

Conclusions: The ratio of TBW to AFM is influenced by body composition (proxy BMI), hydration (proxy CNR), and fluid distribution (proxy wRe/wRi). Therefore, a constant hydration factor of 0.73 should not be used to calculate adipose free mass in HD patients. The best correlation coefficient and smallest SD of bias was obtained using the regression model (eTAT4) based on body mass, age, BMI, wRe and wRi with bioimpedance measurement.

SAP267 Table 1 comparison of TATMRI (25.0 ± 10 kg) with eTAT by four methods

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Healthy Subjects (n=20)</th>
<th>ESRD Patient (n=79)</th>
</tr>
</thead>
<tbody>
<tr>
<td>eTAT (kg)</td>
<td>133±9</td>
<td>138±29</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>83±8</td>
<td>86±16</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>24.5±3.8</td>
<td>26.8±5.2</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>3.8±0.2</td>
<td>3.8±0.49</td>
</tr>
<tr>
<td>PTH (ng/dL)</td>
<td>5.1±1.3</td>
<td>-</td>
</tr>
<tr>
<td>TAC (mg/dL)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>PFT (µL/L)</td>
<td>17±6</td>
<td>17±7</td>
</tr>
<tr>
<td>LDL (mg/dL)</td>
<td>101±21</td>
<td>13±37</td>
</tr>
<tr>
<td>HDL (mg/dL)</td>
<td>36.7±9.3</td>
<td>38±14</td>
</tr>
<tr>
<td>Triglyceride (mg/dL)</td>
<td>128±97</td>
<td>168±94</td>
</tr>
<tr>
<td>Calcium (mg/dL)</td>
<td>-</td>
<td>9.1±0.9</td>
</tr>
<tr>
<td>Phosphorus (mg/dL)</td>
<td>-</td>
<td>4.6±1.3</td>
</tr>
<tr>
<td>hs-CRP (mg/dL)</td>
<td>9.9±3.2</td>
<td>14.9±16.9</td>
</tr>
<tr>
<td>TAC (µg/L)</td>
<td>13.6±6.8</td>
<td>184±48</td>
</tr>
<tr>
<td>CAC (µg/L)</td>
<td>40.0±119.61</td>
<td>126.4±242.95</td>
</tr>
<tr>
<td>FTT (cm²)</td>
<td>11.7±5.7</td>
<td>16.5±11.1</td>
</tr>
</tbody>
</table>

**SAP267 PERI-AORTIC FAT TISSUE AND MALNUTRITION-INFLAMMATION-ATHEROSCLEROSIS/CALCIFICATION SYNDROME IN END-STAGE RENAL DISEASE PATIENTS**

Kultigin Turkmen1, Hatice Kaycioglug2, Ibrahim Guney3, Lutfullah Altintepe2, Orhan Oztek1, 3and Halk Zeki Tombul4

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**Introduction and Aims:** Thoracic peri-aortic fat tissue (PFT) was considered as a metabolically active organ that has a pathogenic role in the genesis of atherosclerosis. Malnutrition, inflammation, atherosclerosis/calcification (MIAC), and endothelial dysfunction are the most commonly encountered risk factors of cardiovascular disease in ESRD patients. We aimed to investigate the relationship between PFT and MIAC syndrome in ESRD patients.

**Methods:** 79 ESRD patients (30 females, 49 males) receiving PD or HD and 20 healthy control subjects enrolled in this cross-sectional study. PFT, thoracic aortic calcification (TAC) and coronary artery calcification (CAC) were performed by a
doi:10.1093/ndt/gfs238 | ii407

**ALT: Alanine amino transferase LDL: Low-density Lipoprotein, HDL: High-density Lipoprotein, hs-CRP: high sensitive C-reactive protein, PTH: Parathyroid hormone, TAC: Toracic-aortic calcification, CAC: Coronary artery calcification, PFT: Peri-aortic fat tissue**

a Comparison with Mann-Whitney test for continuous variables or χ2 test for nominal variables

b Spearman rank univariate analysis for PFT

SAP267 Table 1 The demographic, clinic and laboratory features of the ESRD patients and healthy subjects.
Abstracts

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HIGH DENSITY LIPOPROTEIN VARIABILITY AND LOW TRIGLYCERIDE LEVELS PREDICT MORTALITY IN INCIDENT HEMODIALYSIS PATIENTS BY VIRTUE OF THEIR ASSOCIATION WITH INFLAMMATION

George A Kaysen1, George A Kaysen1, Len A Usvyat2, Stephan Thijssen2, Nathan W Levin3 and Peter Kotanko3

1Department of Medicine Division of Nephrology Uc Davis, 2Renal Research Institute, New York, USA, 3Renal Research Institute, New York USA

Introduction and Aims: The association between high density lipoprotein (HDL) levels and outcomes in dialysis patients is uncertain. Triglyceride levels (TG) are inversely associated with HDL levels and have been associated with cardiovascular risk in patients with chronic kidney disease. This study was conducted to examine the relationship between baseline HDL and TG levels and variability in HDL and TG levels and their association with outcomes among incident hemodialysis patients (HD). Methods: As part of routine clinical practice, we measured HDL and TG in all incident HD patients between 1/2000 and 12/2010. Only patients with 4 or more HDL and TG measurement in the first 365 days and who survived for that period were included. Among those, baseline HDL and TG during year 1 and their variability (including subjects with significant increases or decreases) during the same period were computed using coefficient of variation (CV). The effect of nutrition was measured as normalized protein catabolic rate (nPCR) and of inflammation as neutrophil lymphocyte ratio (NLR). Survival status was analysed as a Cox model during year 2 of HD. Patients were stratified into tertiles of baseline HDL and TG and stratified by variability of HDL and TG by tertiles of CV. Results: We studied 1321 incident HD patients. HDL levels were significantly greater in women (P < 0.001), African Americans (P = 0.001), non-Hispanics (P > 0.001), and patients with no residual renal function (P = 0.001). HDL levels were directly associated with age and pre-HD systolic blood pressure and inversely associated with body mass index (BMI). There was no association between HDL level and mortality. By contrast, high HDL variability (upper tertile, CV > 0.16) in the first year was associated with increased mortality in the following year (P < 0.001). The association with increased mortality included patients with increasing or decreasing HDL levels even when analyzed separately. The effect of variability in HDL was associated with baseline serum albumin and was eliminated by adjustment for baseline albumin. TG was positively associated with albumin, BMI, Hispanic ethnicity, presence of residual renal function and negatively associated with age. African American race and NLR. In contrast to HDL, patients in the lowest TG tertile (< 121 mg/dL) had significantly increased mortality (P = 0.008), but variability in TG had no effect on outcome. The effect of TG level on outcome was no longer significant when adjusted for albumin. NLR but not nPCR was positively associated with HDL variability (P = 0.01) but not with HDL levels, and negatively associated with TG levels (P < 0.05) but not with TG variability. Conclusions: HDL cholesterol levels have no effect on outcome in HD patients who survive the first year of treatment. Factors that change HDL levels and/or increase variability do play a strong role in outcome. Of these, it is most likely factors that determine albumin level, most likely inflammation measured here as NLR, that determine both HDL variability and patient outcome. Similarly, the relationship between low TG level and outcome was entirely associated with albumin. Thus inflammation and its association with albumin levels dominate the roles of individual lipoproteins, the stability of HDL levels and the absolute level of TGs in determining their association with mortality of prevalent HD patients. Thus lipoprotein levels serve as surrogate measures of inflammation in determining their effect on mortality.

SAP270

ACTIVATION OF NUCLEAR FACTOR-K B IN THE AORTA OF PATIENTS IS ASSOCIATED WITH THE SEVERITY OF ATHEROSCLEROSIS BUT NOT WITH CHRONIC KIDNEY DISEASE.

Miguel Hueso1, Joan Torras1, Marta Carrera1, August Vidal1, Estanis Navarro2, Israel Rivas1, Ines Ramal1, Nuria Bolaños1, Cristian Varela2, Alberto Martinez-Castaño1 and Josep M. Ginyo1

1Hospital Bellvitge-IDIBELL, 2Laboratori D’oncologia Molecular-IDIBELL, 3Idibell

Introduction and Aims: Chronic kidney disease (CKD) is associated with a high incidence of atherosclerosis (AE) and cardiovascular disease (CVD). This fact cannot be fully explained by traditional risk factors for AE, suggesting that other factors, as oxidative stress, uremic toxins or inflammation, are involved in AE development in CKD patients. In inflammatory conditions, nuclear factor-k B (NF-kB) has been found to be frequently activated, and consequently, activated NF-kB has been detected in human AE. In this work, we aimed to investigate the changes of NF-kB in the aorta of patients with CKD. Methods: This is a case-control study in patients who died in our hospital and whose autopsy was authorised. Cases (n = 5) were patients with CKD in stage 3, 4 or 5 before the admission into the hospital. Controls (n = 5) included patients with similar characteristics, but with a normal glomerular filtration (eGFR) estimated by MDRD-IDMS. Samples from 3 different areas of the aorta were obtained: one from a macroscopically normal aorta, a second one from an area with incipient atherosclerotic lesion, and the third sample from a complicated lesion. Samples were fixed in formalin and embedded in paraffin for immunohistological analysis. Infiltrating mononuclear cells were identified using the S-100 (dendritic cell) and CD3 (T-lymphocyte) markers. Presence of NF-kB was detected with an anti-p65-specific antibody. Immunostaining intensity was scored semi-quantitatively (0 = no staining, 1 = weak staining, 2 = moderate staining, and 3 = strong staining). Results: Severity of AE lesions was associated with an increase of infiltrating dendritic cells in the intimae (p = 0.07), and T lymphocytes in the adventitia tunicae (p = 0.05). Activated NF-kB was detected mainly in vascular smooth muscle cells. NF-kB expression was increased both in the intimae (p = 0.042) and in adventitia (p = 0.057) according with the severity of the lesions. However, no differences were
observed either in inflammatory infiltrate or in the expression of NF-kB in different stages of AE, according to the presence of CKD.

**Conclusions:** Our study detected activation of NF-kB in AE lesions but failed to show any association with CKD. This indicates that CKD vascular microenvironment would not be associated with the overall arterial inflammatory status, and that the high incidence of AE in CKD patients would be related to other factors.

**SAP271**

**THE EFFECT OF N-3 POLYSATURATED FATTY ACIDS ON ADIPONECTIN LEVELS IN PATIENTS WITH END STAGE RENAL DISEASE**

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**Introduction and Aims:** In subjects without kidney disease, adiponectin appears to have beneficial anti-diabetic, anti-thrombogenic and anti-atherogenic effects. Also, in patients with end stage renal disease (ESRD) high levels of plasma adiponectin are associated with a reduced risk of cardiovascular disease. n-3 polysaturated fatty acids (PUFA) have several beneficial effects in ESRD patients and the aim of the present study was to assess the effect of n-3 PUFA supplementation on adiponectin levels in ESRD patients.

**Methods:** In a double blinded intervention trial 162 ESRD patients (mean age 67 years; n = 35, 36 women and 106 men) undergoing chronic haemodialysis was randomized to a n-3 PPUA daily or placebo for 3 months. Adiponectin and lipids and lipoproteins were measured at baseline and at the end of the intervention period. 

**Results:** As baseline a correlation was found between adiponectin and HDL-cholesterol (r = 0.531, p < 0.01), and an inverse correlation between adiponectin and body mass index (r = -0.43, p < 0.001). In the n-3 PPUA group baseline adiponectin was 18.5 μg/ml; +11 and after 3 months of supplementation no significant change was observed (18.8 μg/ml; +12, P = 0.56). Also compared to placebo n-3 PPUA supplementation did not change adiponectin levels.

**Conclusions:** Administration of n-3 PUFA has no effect on adiponectin levels in ESRD patients. However, as observed in other populations adiponectin is closely associated with body mass index and HDL-cholesterol in ESRD patients.

**SAP272**

**PROCALCITONIN IS NOT SUPERIOR TO C-REACTIVE PROTEIN AS A MARKER OF INFECTION IN PATIENTS WITH CHRONIC KIDNEY DISEASE**

Ji Hyeon Park1, Eun Hee Koo1, Hyun Kyung Kim1, Min Su Kim1, A Jin Cho1, Jung Eun Lee1, Hye Ryoun Jung1, Wooseong Huh1, Dae Joong Kim1, Yoon-Goo Kim1 and Ha Young Oh1
1Samsung Medical Center, Seoul, Korea

**Introduction and Aims:** Recently, procalcitonin (PCT) is used as a surrogate marker for predicting infection in chronic kidney disease (CKD) patients because nonspecific elevation of C-reactive protein (CRP) caused from chronic inflammation was reported in these patients. However, it is uncertain whether PCT is more accurate or cost effective for detecting infection compared to CRP in CKD patients. We investigated the clinical usefulness of PCT and CRP in patients with CKD state 1-5.

**Methods:** From November 2008 to July 2011, a total of 423 patients who underwent PCT and CRP tests simultaneously at SMC were included. Infection was defined when clinic signs of systemic inflammatory response were present, determined by a definable source of infection (microbiology confirmed) and/or positive blood cultures. In the group I, a total of 244 patients were in infection. Patients had CKD stage 1,2,3,4, or 5 (n = 36, 7, 39, 33, 109). The 109 CKD stage 5 patients in the group I were divided into three subgroups: no dialysis (n = 38), hemodialysis (HD) (n = 36), and peritoneal dialysis (PD) (n = 35). In the group II, a total of 199 patients were without infection. Patients had CKD stage 1,2,3,4, or 5 (n = 332, 38, 32, 84). The 84 CKD stage 5 patients in the group II were divided into three subgroups: no dialysis (n = 35), HD (n = 36) and PD (n = 15).

**Results:** Both serum PCT and CRP levels were significantly high in infection group compared to non-infection group (Table 2). The area under the receiver operating characteristic (ROC) curve in the diagnosis of infection versus non-infection was 0.844 for PCT and 0.880 for CRP (Figure 1A). In CKD stage 1-5, the area under ROC curve was 0.881 for CRP and 0.877 for PCT (Figure 1B). In the group II, PCT increased in parallel to deterioration of eGFR (r = 0.226, p = 0.01). Table 1. Baseline characteristics

**Table 1. Baseline characteristics**

**Table 2.** Baseline characteristics of patients with and without infection.

**Table 3.** Disease categories in group I. B. Disease categories in group II. Fig1. Receiver operating characteristic (ROC) curve for prediction of infection A. In CKD stage 1-5, area under ROC curve in the diagnosis of infection versus non-infection was 0.844 for procalcitonin (PCT) (red line) and 0.844 for C-reactive Protein (CRP) (blue line). B. In CKD 3A, 3C, and 5 area under ROC curves was 0.887 for PCT (red line) and 0.881 for CRP (blue line). Fig2. The correlation between procalcitonin (PCT) and C-reactive protein (CRP) A. In group I, PCT and CRP showed positive correlation (r = 0.517, p-value < 0.01). B. In group II, PCT and CRP showed positive correlation (r = 0.367, p-value < 0.01).

**Conclusions:** Our study demonstrates that PCT is still accurate and cost effective for predicting infection in CKD patients. PCT may not superior to CRP in regard to both diagnosis and cost benefit in CKD patients.
also after stimulation with fMLP. Adalimumab partially inhibited the stimulating effects of TNFa (TNFa: 16.2% vs. control: 7.6%, P < 0.05; TNFa: 16.2% vs. TNFa; +; Adalimumab: 11.1%, not significant; control: 7.6% vs. TNFa; +; Adalimumab: 11.1%, not significant). Similar results were observed for synergistic effect of TNFa and IL1 on free radical production both in monocytes and granulocytes. In contrast, IL6, IL18 and IL1β did not have an effect on ROS-production at concentrations observed in CKD.

**Conclusions:** Our data indicate that known cytokines are not necessarily pro-inflammatory in concentrations as occurring in CKD. Only TNFa stimulates monocytes in basal condition and after stimulation with fMLP, while others like IL6, IL18 and IL1β do not induce free radical production in leukocytes in a broad 100-1000 concentration range. The basal pro-inflammatory effect of TNFa can be entirely blocked by Adalimumab, while Adalimumab inhibits only partially ROS-production in activated leukocytes.

**SAP276**

**USE OF ALLOPURINOL IS ASSOCIATED WITH REDUCED ARTERIAL STIFFNESS IN PROGRESSIVE CHRONIC KIDNEY DISEASE (CKD)**

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**Introduction and Aims:** CKD is associated with heightened oxidative stress and increased arterial stiffness. Hyperuricaemia, which is prevalent among CKD population, is known to be associated with adverse cardiovascular outcome. Allopurinol, which is widely used for treatment of hyperuricaemia and gout, has recently been shown to improve left ventricular mass and endothelial dysfunction in CKD. We hypothesized that the use of allopurinol is associated with reduced arterial stiffness in patients with progressive CKD.

**Methods:** Renal Impairment in Secondary Care (RISC) is an on-going prospective observational study, aiming to recruit 1000 pre-dialysis patients with progressive CKD and follow-up for 10 years. Arterial stiffness is measured non-invasively by carotid-femoral pulse wave velocity (PWV) using Vicorder system.

**Results:** 211 patients (mean age: 61.4 ± 16.6 years, 60.2% male) were recruited to date. Mean GFR and ACR were 26 ± 12 ml/min and 121.7 ± 162.1 mg/mmol. 14.7% of patients had background of cardiovascular disease (CVD), 17.1% did not have an effect on arterial stiffness in patients with progressive CKD.

**Conclusions:** Use of allopurinol is associated with reduced arterial stiffness in CKD. We hypothesized that the use of allopurinol is associated with reduced arterial stiffness in patients with progressive CKD.

**SAP276**

**INSULIN GROWTH FACTOR-1 (IGF-1) AS A PREDICTOR OF NUTRITIONAL STATUS AND MORTALITY IN CHRONIC KIDNEY DISEASE (CKD) STAGE 5 PATIENTS**

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**Introduction and Aims:** Abnormalities in the IGF-1/growth hormone system contribute to catabalism in CKD in whom the bioavailability of IGF-1 is decreased. We investigated factors associated with changes of IGF-1 levels in CKD stage 5 patients during their first year of dialysis treatment, and the predictive power of IGF-1 after 48 months of follow-up.

**Methods:** In 207 CKD stage 5 patients (median GFR 7 ml/min, median age 56 years, mean 61%), fasting plasma samples were obtained both before and after one year of dialysis treatment for measurements of plasma IGF-1 (by a chemiluminescent immunometric assay) and C-reactive protein (CRP). Nutritional status was assessed by subjective global assessment (SGA) and bone mineral density (BMD) was measured by DXA. Associations between levels of IGF-1, CRP, BMD, nutritional status and mortality were evaluated by means of Kaplan Meier curves and Cox proportional hazards model.

**Results:** During the first year of dialysis treatment, the median concentration of IGF-1 increased in both patients treated with peritoneal dialysis (PD) and hemodialysis (HD), from 181 (87-320) to 240 (113-413) ng/ml (p < 0.05), with no significant difference between dialysis modalities. Whereas IGF-1 was significantly lower in diabetic (DM) patients compared with non-DM patients, before dialysis and after one year of dialysis therapy, the degree of improvement of IGF-1 concentrations during the first year of dialysis did not differ between DM and non-DM patients. IGF-1 did not differ between inflamed patients (CRP; < 10 mg/L) vs. non-inflamed patients, neither before start of dialysis, nor after one year of dialysis. IGF-1 correlated inversely (rho= -0.18; p < 0.05) with signs of poor nutritional status (SGA score; < 1) before dialysis treatment, but this association was lost after one year of dialysis (rho= -0.12; p = 0.09). IGF-1 correlated positively with BMD both before dialysis and after one year of dialysis. Whereas PTH was not associated with IGF-1 (at baseline or after one year of dialysis), baseline PTH was the only independent, albeit weak, predictor of the change in IGF levels during the first year of dialysis (r2=0.05; p < 0.05). A low IGF-1 level was associated with higher mortality risk in univariate analysis, both before and after one year of dialysis, and in multivariate Cox analysis, after adjusting for age, gender, DM, CVD and SGA, it remained as an independent risk factor for mortality in the patients who had been on dialysis for one year (Figure 1).

**Conclusions:** As PTH was the only factor associated with the increase of IGF-1 levels during the first year of dialysis treatment this suggests interplay between these two hormones. A low level of IGF-1 is an independent predictor of mortality in patients undergoing dialysis.
cardiovascular event. Patients with persistently elevated IL-6 values had higher risk to develop cardiovascular events (OR: 1.21 (1.11-1.32), p = 0.001). Mean of two measurements of IL-6 was better predictor for events than a single measurement of IL-6, CRP, TNF-α and IL-18. Patients who had hypertension and increased plasma IL-6 greater than 6.6 pg/ml and those with previous peripheral vascular disease had an increase risk for cardiovascular events (2.3, 1.05-5.22, p = 0.037 and 2.95 (1.27-6.93), p <0.011, respectively) in an adjusted Cox regression model.

Conclusions: IL-6 is more inflammatory marker than TNF-α and IL-18 in predicting cardiovascular events in CKD non-dialysis patients. Mean of two measurements is better than simple determinations at predicting cardiovascular outcome. Moreover, persistently elevated IL-6 values after taking statins or angiotensin II receptor blockers predicts cardiovascular events in CKD patients.

Introduction and Aims: We have been demonstrated that the impact of oxidized high-density lipoprotein (oxHDL), dysfunctional HDL, on mortality and cardiovascular disease (CVD) events in patients with CKD 5d (Atherosclerosis 2011 in press). The aim of this study was to assess the association of oxHDL, subfraction of oxHDL, with CKD stage.

Methods: This cross-sectional study examined a cohort of patients with CKD (n = 106). Blood samples were obtained to measure creatinine, total cholesterol (TC), HDL-C, low-density lipoprotein (LDL-C), high sensitive-C reactive protein (hsCRP), apolipoprotein (ApoA1, ApoA2, ApoB),subfraction of HDL (HDL2, HDL3), oxidized LDL, oxHDL and subfraction of oxHDL (oxHDL2, oxHDL3). Oxidized HDL and subfraction of oxHDL were assessed by Elfisa using monoclonal anti-oxidized ApoA1 antibody.

Results: Patients were grouped according to CKD stage (stage 2-3 n = 30, stage 4 n = 27, stage 5 n = 24, stage 5d n = 25). Levels of LDL-C, ApoA1, ApoA2, ApoB and HDL3 were decreased according to severity of CKD stage, however, HDL-C and HDL2 levels did not differ among patients with different stages of CKD. Whereas severity of CKD stage was associated with low levels of ApoA1 (ApoA1-HDL3) and ApoA2 (ApoA2-HDL3) in HDL3, levels of ApoA1 in HDL2 (ApoA1-HDL2) were similar among patients with different stages of CKD. Levels of oxHDL and oxHDL3 were decreased especially in patients with CKD 5d, however, oxHDL2 levels were elevated in those patients. Advanced CKD was associated with high ratios of oxHDL/ApoA1 and oxHDL2/ApoA1-HDL2, but not with oxHDL3/ApoA1-HDL3 ratio.

Conclusions: Decrease in GFR in patients with CKD is associated with increase in oxHDL2, oxidized anti-atherogenic lipoprotein, and this may be one of the reasons for advancing atherosclerosis or high prevalence of CVD events in CKD patients.

Introduction and Aims: Nephrotic syndrome (NS) is associated with increased risk of cardiovascular diseases and arterial stiffness is associated with cardiovascular events. However, there is no any data in the literature up-to-date on arterial stiffness in patients with NS. Thus, in this study, we aimed to evaluate arterial stiffness by the carotid-femoral pulse wave velocity measurements along with volume and nutritional parameters by bio-impedance measurement, in patients with NS in comparison with a healthy control group.

Methods: Thirty-four adult patients with newly diagnosed and not treated NS and 34 healthy control enrolled in this study. Arterial stiffness was assessed by carotid-femoral pulse wave velocity (C-f PWV), in patients with NS in comparison with healthy controls.

Results: Mean age of the patients was 44.6±18.7 years (18-72). Mean carotid-femoral pulse wave velocity (C-f PWV) value was 8.3±2.5 m/s in patients with NS and 6.7±1.1 m/s in the control group (p = 0.002). In univariate analysis, C-f PWV was positively correlated with age, systolic blood pressure, mean arterial pressure, pulse pressure, body mass index, body fat ratio, waist - hip ratio, creatinine and uric acid and negatively correlated with creatinine clearance. In the linear regression analysis, it was observed that age and MAP data predicts the C-f PWV. There was no difference in terms of the PWV values of the patients according to renal biopsy diagnosis. Total body fluid, extracellular fluid volume, ECW/Height, ECW/BWSA, third space volumes was higher and the fatty tissue ratio was lower in patients with NS.

Conclusions: In this cross-sectional study we report that that the patients with NS have increased arterial stiffness and are more hypervolemic compared to the healthy subjects. As a result, we suggest that in the pathogenesis of the increased arterial stiffness in patients with nephrotic syndrome the age, increased oxidative stress, hypervolemia, and hypertension plays a role.
apo A-I, apo B, Ox-LDL and TC/HDLc ratio, were also observed. HDLc levels were also lower for patients but this result did not reach statistical significance (p = 0.082).

Similar changes were observed for patients with or without statin therapy, as compared to controls, except for Lp(a). Multiple linear regression analysis, showed that BMI, HDLc, time on hemodialysis and TG were independent determinants of adiponectin levels, and that apo B, TG and LDL-cholesterol, were independent determinants of Ox-LDL concentration. Concerning the apo(a) genotype, the homozygous (TTTTA)8/8 repeats was the most prevalent (50.8%), followed by the heterozygous (TTTTA)8/9 (17.1%) and (TTTTA)8/10 (15.5%) repeats. Lp(a) levels did not present a clear tendency among different genotypes. Moreover, we divided our patients in two groups, using the traditional cut-off for an atherothrombotic Lp(a) concentration (30.0 mg/dL) and for a cut-off corresponding to the median value found in our patients (44.9 mg/dL). The distribution of the alleles was significantly different (p = 0.030) according to the median cut-off for our groups.

Conclusions: HD patients presented, mainly, qualitative risk changes in lipid profile, rather than quantitative changes (e.g. significant decrease in apo A). Actually, a raised proportion of LDL particles that are oxidized was observed. Adiponectin almost doubled its values in patients and seems to be an important determinant in HDLc and TG levels, improving the lipid profile in these patients. Apo (a) alleles with a lower number of repetitions are more frequent in patients with higher Lp(a); even though, no association was found between apo (a) genotypes and Lp(a) concentration. Plasmatic factors, related to CKD or to HD procedure, may be involved in Lp(a) metabolism in HD patients, which may mask the association between the PNRP in the apo(a) gene and Lp(a) levels.

SAP282

**EFFECT OF DARK CHOCOLATE INTAKE ON RENAL TISSUE OXYGENATION AS MEASURED BY BOLD-MRI**

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Introduction and Aims: Dark chocolate is rich in flavanoids, has anti-oxydative properties and increases the bioavailability of NO. As such, consumption of dark chocolate has been reported to decrease blood pressure and induce coronary vasodilatation, yet its effects on renal hemodynamics are largely unexplored. Adequate renal tissue oxygenation is crucial for the maintenance of renal function and depends of renal perfusion and local oxygen consumption. The goal of this study was to investigate the effect of dark chocolate on renal tissue oxygenation in humans, as compared to flavanoid-poor white chocolate.

Methods: Eight healthy volunteers with preserved kidney function (mean age ± SD 37 ± 11y, 62.5% women, BMI 21 ± 3 kg/m2, eGFR 104 ± 14 ml/min/1.73m2) underwent blood oxygenation-level dependent magnetic resonance imaging (BOLD-MRI) before and two hours after the ingestion of 1g/kg of dark chocolate (70% cocoa). Four participants were tested at least one week later before and two hours after the ingestion of 1g/kg of dark chocolate.

Eight healthy volunteers with preserved kidney function (mean age ± SD 37 ± 11y, 62.5% women, BMI 21 ± 3 kg/m2, eGFR 104 ± 14 ml/min/1.73m2) underwent blood oxygenation-level dependent magnetic resonance imaging (BOLD-MRI) before and two hours after the ingestion of 1g/kg of dark chocolate (70% cocoa). Four participants were tested at least one week later before and two hours after the ingestion of 1g/kg of dark chocolate. Four coronal slices were selected in each kidney, and combination sequence was used to acquire T2* weighted images. The mean R2* values (=1/T2*) were calculated, a low R2* indicating a high tissue concentration.

Results: BP did not change after chocolate intake. The mean medullary R2* was lower after dark chocolate intake compared to baseline (29.5 ± 0.13 s-1 vs 28.3; ± 1.4 s-1, P = 0.09), indicating a trend towards higher medullary oxygenation after dark chocolate intake in all but one participant (figure 1), whereas medullary oxygenation slightly decreased after white chocolate intake (27.5 ± 2.1 vs 27.9 ± 1.7, p = 0.23, figure 2). There were no significant changes in cortical oxygenation after black or white chocolate intake.

Conclusions: This pilot study suggests for the first time an acute effect of dark chocolate on renal medullary oxygenation. Whether this is linked to flavanoid-induced changes in renal perfusion or oxygen consumption, and whether chocolate has potentially renoprotective properties merits further study.

SAP283

**AN ORIGINAL MODEL FOR DIAGNOSIS AND FOLLOW-UP OF MALNUTRITION IN CHRONIC KIDNEY DISEASE PATIENTS**

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Introduction and Aims: Protein-energy malnutrition (m) in outpatients with CKD is an extremely important problem but is not well recognized and the criteria for diagnosis are arbitrary and not universal. The purpose of this study was to evaluate in patients (p) with different degree of CKD the prevalence of m and inflammation and their modifications in the time using a novel score derived from standard nutritional parameters.

Methods: 161 p with CKD and free diet were enrolled and divided according to GFR calculated from MDRD: stage II, n = 23 (M 15 F 8 mean age 60 ± 19 yrs); stage III, n = 49 (M 45 F 24 mean age 63 ± 15 yrs); stage IV, n = 57 (M 36 F 21 mean age 71 ± 16 yrs); stage V, n = 11 (M 7 F 4 mean age 70 ± 10 yrs). For each p we considered: age, heart disease, C-reactive protein (CRP), albumin, total lymphocytes, total cholesterol, Body Max Index (BMI), Subjective Global Assessment (SGA) and analysis of body composition derived from Bioelectrical Impedance Analysis: PA=Phase Angle, FM=Fat mass, FFM=Free Fat Mass, BCMi=Body Cell Mass, BCMci=Body Cell Mass Index. The score used in this study is shown in table. 1. P were followed-up from at least 1 to 3 years.

Results: At baseline, according to m score, the prevalence of m was different in regard to the stage renal disease: Score= 0: stage II= 35%, stage III= 20%, stage IV = 2%, stage V = 0%; Score = 1: stage II= 43%, stage III = 32%, stage IV = 19%, stage V = 9%; Score = 2: stage II = 9%, stage III = 15%, stage IV = 18%, stage V = 9%; Score = 3: stage II = 4%, stage III = 16%, stage IV = 26%, stage V = 36%; Score = 4: stage II = 9%, stage III = 17%, stage IV = 35%, stage V = 46%. The percentage of subjects with CRP < 1 were: stage II 13%, stage III 14%, stage IV 28%, stage V 36% with an high prevalence of cardiovascular disease. At the end of the follow up a significant worsening of m was observed: the prevalence of p with normal nutritional status and high prevalence of p with light, moderate and severe m (p < .01) over time.

Conclusions: Our results confirm the high prevalence of m in outpatients with CKD, mainly in stage IV-V with a high percentage of p at risk of m in stage II (43%). Without specific nutritional intervention, m is getting worse in time; the high prevalence of inflammation is correlated with the degree of m; BIA represents an attractive clinical tool to detect impairment of body composition from the early stages of CKD; our m score has proved to be an useful, simple and valid tool to identify not only malnourished patients but also those at risk of m.
NUTRITIONAL PARAMETERS pathological values (NP) | CRITERIA DIAGNOSIS OF MALNUTRITION | MALNUTRITION SCORE
--- | --- | ---
SGA | > 0 | RISK OF MALNUTRITION: SGA < 0 NP | 0 ABSENT
ALBUMIN | < 3.5 g/dL | LIGHT: SGA + 1 NP | 1 RISK OF MALNUTRITION
LYMPHOCYTE | < 1800/mm^3 | MODERATE: SGA + 2 NP | 2 LIGHT
CHOLESTEROL | < 100 mg/dL | SEVERE: SGA + 3 NP | 3 MODERATE
BMI | < 23 | 4 SEVERE
PA | < 4.8 | \nFM | < 10% | \nFFM | ↓ 5 % in 3 months | \nBCM | M < 40% | \nBMI | < 17 | \n
**SAP284**

**TREATMENT OF CHRONIC HCV WITH PEGYLATED IFN ALPHA 2-A IN HEMODIALYSIS PATIENTS; SINGLE SAUDI CENTER EXPERIENCE**

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**Introduction and Aims:** Hepatitis C virus (HCV) infection is a global problem and is common worldwide. According to the World Health Organization (WHO) data about 3% of the general population is infected by HCV; this indicating that 200-300 million individuals may be affected. One million new cases of infection are reported annually. After kidney transplantation, recipients with HCV have an increased risk of mortality and chronic liver disease compared with those without HCV. Therefore, treatment of HCV with interferon after kidney transplantation may increase the risk of graft rejection. In this study we will investigate the tolerability and efficacy of pegylated interferon alpha -2b with Ribavirin in special group of patients.

**Methods:** The present study was carried out in Prince Salman Center for Kidney Diseases, Riyadh, Saudi Arabia. The 14 participant patients were underwent chronic HD treatment for ESRD during the study period. Hemodialysis was carried out routinely three times a week. Treatment schedule Pegylated interferon fourteen patients were treated with PEGINTRONTM (peginterferon alfa-2b - 12 KD Schering- plough) at a dose of 1mg/kg once weekly subcutaneously following HD. Treatment was continued for a total duration of 48 weeks Ribavirin therapy This was added to peginterferon alfa-2b in a dose of 200 mg given every other day following the HD session. And Ribavirin was stopped when Bilirubin-indirect level; < 5MG/DL (or; < 85.5 umol/L) Primary outcomes The primary outcomes were sustained viral response and serious adverse events. Sustained virological response (SVR) was defined as undetectable hepatitis C RNA by PCR in the serum 12 weeks after stopping therapy. Serious adverse events were defined as those resulting in hospitalization, death or any intervention not part of routine standard of care. Primary virological response (PVR) was defined as undetectable hepatitis C RNA by PCR in the serum 12 weeks after starting treatment or two log decreases in the viral titer, and end of treatment response defined as undetectable serum HCV RNA by PCR.

**Results:** Our study included 14 ESRD patients with HCV infection. (Ten men and four women), their mean age was 46.28 ± 25.72 years, and their mean HD duration was 35.7 months. Their virological response Twelve weeks after starting treatment, one patient could not tolerate treatment, quantitative HCV –RNA PCR was done, three patients (21.4%) were still positive. And in the remaining ten patients nine were HCV- RNA PCR negative, and one patient had two log decreases in the viral titer, and Primary virological response PVR was (71.4%). After forty-eight weeks of treatment HCV –RNA PCR qualitative and quantitative were tested for ten patients and all the results were negative (71.4%). Twenty-four weeks after stoppage of treatment HCV –RNA PCR qualitative and quantitative were tested again for the ten patients and results were negative for nine patients and positive for one patient and SVR was (64%).

**Conclusions:** Our data show that treatment of chronic HCV infection in dialysis patients with peginterferon alpha -2b at a dose of up to 1mg/ kg/week with Ribavirin is well tolerated and safe in dialysis patients. Furthermore, SVR was achieved in 64% of patients. So, peginterferon alpha -2b is likely to become a valuable addition for HCV therapy in ESRD when combined with reduced Ribavirin doses.

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**SAP285**

**INCREASED LEVELS OF SERUM AMYLOID A IN END STAGE RENAL DISEASE PATIENTS IS ASSOCIATED WITH THE PRESENCE OF A UNIQUE BIOMARKER IDENTIFIED BY PROTEIN CHIP ARRAY ANALYSIS**

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**Introduction and Aims:** Serum amyloid A (SAA) is an acute phase reactant which is regulated by IL1, IL-6 and TNF. The circulating levels of serum amyloid A are markedly increased in inflammatory conditions. SAA is a 104 amino acid polypeptide with a molecular mass of 12-14 KDa. Protein Chip Array methods have been used to identify unique biomarkers in ESRD patients. The purpose of this study was to determine the relationship of a previously reported biomarker in ESRD with circulating levels of SAA.

**Methods:** Plasma samples from 117 ESRD patients, over the age of 18 on maintenance hemodialysis and 50 age matched controls (healthy volunteers) were included in this study. SAA levels were measured by using a sandwich ELISA method (Abzyme, Needham, MA). Protein Chip Array profiling was carried out utilizing surface enhanced laser desorption (SELDI; BioRad Corp, Hercules, CA) utilizing gold chips.

**Results:** The plasma SAA levels were markedly elevated in ESRD (41.8 ± 12.6; range 4.6-98.1 g/ml) in contrast to the normals (4.5 ± 1.2; range 1.1-9.8 g/ml). SELDI analysis revealed a unique biomarker signal in 78 out of 117 (67 %) in the ESRD patients and 2 out of 50 (4%) in the normal individuals. The presence of the 11.6 KDa biomarker correlated with the high SAA levels.

**Conclusions:** These studies suggest that SAA level upregulation is associated with the presence of the 11.6 KDa biomarker. Protein Chip Array profiling may be useful in the risk stratification of ESRD patients.