

Results: mean age was 58.62 years (range 29-86 years); at the time of diagnosis, eleven patients presented with lumbar pain, 3 with diffuse abdominal pain. Notably, in 2 patients renal infarction was painless. Associated symptoms included macroscopic haematuria (3 patients), arterial hypertension (9), hyperthermia (4), oliguria (2) and dysuria (1). Possible associated risk factors included obesity/overweight (6 patients), current smoking (3), cocaine abuse (1), estroprogestinic therapy (2), atrial fibrillation (4), and atrial mixoma (1 patient). Six out of 11 patients were found to have antiphospholipid antibodies at the diagnosis (anti- β 2GPI antibodies in 3, anticardiolipin antibodies in 2, LAC in 1), but only one had these antibodies still detectable 12 weeks later. Systolic arterial pressure was 147.69 ± 18.99 mmHg, diastolic 83 ± 10 mmHg. Laboratory investigations at onset revealed leukocytosis in 10 patients, elevated lactate dehydrogenase levels in 13 and augmented C-reactive protein in 10. Mean serum creatinine was 1.4 ± 0.6 mg/dL (eGFR 65 ± 30 mL/min/1.73 m²). Acute kidney injury occurred in two patients at presentation. CT scan/MRI imaging showed alterations of renal arteries in 9 cases (thrombosis in 5 patients, renal artery dissection in 1, fibromuscular dysplasia in 2 and both thrombosis and dissection in 1). Thirteen patients were treated with LMWH, 2 with aspirin alone; one patient did not receive anticoagulant/antiplatelet treatment because of severe arterial hypertension. At the end of follow-up (23.6 \pm 36 months in 9 patients), serum creatinine was 1.1 ± 0.2 mg/dL; one patient remained on chronic hemodialysis. In 4/6 patients who underwent renal scintigraphy after a median of 28 months (range 6-120), the contribution of the affected kidney to total renal function was reduced.

Conclusions: 50% of our patients had an idiopathic renal infarction. Renal artery abnormalities (fibromuscular dysplasia, renal artery dissection) were present in 25% of patients, cardioembolic etiology in 25%. Clinical presentation was confirmed to be non specific.

Further studies should focus on etiology and evolution of kidney function in patients with acute renal infarction.

SP150 BIOMARKERS OF EARLY RENAL TUBULAR DAMAGE IN PATIENTS WITH GOUT

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Introduction and Aims: Many patients with gout can have CKD at a later stage, as there are no clinical manifestations for a long time. In this connection it is important to study biomarkers of kidney damage at the preclinical stage of CKD. The aim of the study: to determine level of kidney damage markers in patients with gout without clinical manifestations of CKD.

Methods: We examined 33 gouty patients with normal GFR, with normal microalbuminuria level and kidney ultrasound, thus without any CKD evidence. The average age of the patients was 52 ± 9.4 years, GFR 92.2 mL/min/1.73 m² [85; 101.8]. The control group consisted of 25 healthy volunteers. The average age of them was 47.9 ± 5.6 years, GFR 91.2 mL/min/1.73 m²[88.7; 111.6]. The examination included the detection by ELISA, VEGF, TGF- β 1, KIM-1. Statistical analysis was carried out using a standard package Statistics 6.0.

Results: The group of patients had significantly lower levels of a marker of angiogenesis VEGF-A compared with healthy volunteers: 97 pg/ml [64.6; 262.8] vs 193.3 pg/ml [113.8; 275.6], $p < 0.05$. TGF- β 1 as a marker of fibrosis in patients with gout was significantly lower than in the control group 529.5 pg/ml [478.6, 546.5] vs 549.8 pg/ml [526; 621.5], $p < 0.05$.

Conclusions: Patients with gout without clinical manifestations of CKD showed decrease of the VEGF, TGF- β 1 levels which can indicate the presence of early signs of endothelial dysfunction and renal tubular fibrosis. At the same time a marker of acute tubule injury in patients with gout did not change.

SP151 FIBROBLAST GROWTH FACTOR 23 CORRELATES WITH AORTIC VALVE CALCIFICATIONS AND NON-DIPPER STATUS IN CHRONIC KIDNEY DISEASE PATIENTS

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Introduction and Aims: The aim of the present study is to evaluate the association between fibroblast growth factor 23 (FGF-23) with respect to aortic valve calcifications, arterial stiffness and dipping status in chronic kidney disease (CKD) patients.

Methods: We enrolled 54 subjects: 44 CKD patients in pre-dialysis (8 patients with CKD stage 2, 24 patients with CKD stage 3 and 12 patients with CKD stage 4), and 10 healthy controls. FGF-23, IL-6, TNF α and intact parathyroid hormone (iPTH) were evaluated using xMAP technology (Luminex[®] 200[™]). Echocardiography 2-D and M-mode was used to assess the presence of aortic valve calcifications. Peripheral pulse wave analysis and pulse wave velocity (PWV) was performed using SphygmoCor

device. All patients underwent 24 hour ambulatory blood pressure monitoring (ABPM). Statistical analysis was performed using IBM SPSS Statistics Version 21.

Results: Levels of FGF-23, IL-6 and TNF- α and were significantly higher ($p < 0.05$) in CKD patients compared to healthy controls. The levels of FGF-23, IL-6 and TNF- α inversely correlated with estimated glomerular filtration rate. Aortic valve calcifications were present in 58.2% patients, left ventricular hypertrophy in 72.2%, and 54.4% patients had non-dipper pattern. Mean values for stiffness parameters were: PWV = 9.87 ± 2.8 m/sec, left ventricular ejection duration index (ED) = 32.8 ± 3.3 %, subendocardial viability ratio (SEVR) = 159.8 ± 26.3 %. FGF-23 ($p = 0.01$), TNF- α ($p = 0.015$) and eGFR ($p = 0.008$) significantly correlated with aortic valve calcifications. FGF-23 significantly correlated with markers of inflammation and mineral disturbances (TNF- α , $p = 0.01$; IL-6, $p = 0.0001$; iPTH, $p = 0.01$), and proteinuria ($p = 0.008$). Also, FGF-23 correlated with ED ($p = 0.02$), and SEVR ($p = 0.019$), but not with PWV. FGF-23 and IL-6 were significantly increased in CKD patients with non-dipper versus dipper status ($p < 0.05$).

Conclusions: Our results demonstrated a link between FGF-23, aortic valve calcifications, left ventricular function (evaluated by ED and SEVR) and non-dipper status in chronic kidney disease patients.

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SP152 SERUM OMENTIN-1 LEVELS IN DIABETIC AND NONDIABETIC PATIENTS WITH CHRONIC KIDNEY DISEASE

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Introduction and Aims: Omentin-1, a novel adipokine identified in visceral adipose tissue, is negatively correlated with different conditions such as diabetes, obesity and inflammation. However, changes in serum Omentin levels associated with the degree of the renal dysfunction and with metabolic risk factors in Chronic Kidney Disease (CKD) patients has not yet been revealed. In the present study, we aimed to investigate the level of Omentin-1 and related parameters in diabetic and nondiabetic CKD patients.

Methods: Sixty-four (30 diabetic, 34 non-diabetic) CKD patients and 27 healthy control subjects enrolled in this cross-sectional study. Patients with conditions that possibly affect serum Omentin levels were excluded. Serum levels of albumin and C-Reactive Protein (CRP) were used to determine malnutrition, and inflammation, respectively, just as in studies evaluating high cardiovascular mortality and morbidity in CKD patients. Malnutrition defined as serum albumin < 3.5 mg/dL and inflammation was defined as serum CRP level of > 10 mg/L (normal range, 0-5 mg/L). The patients were classified as malnutrition-inflammation (MI)-0 (no component), MI-1 (one component) and MI-2 (two components). Anthropometric and laboratory assessment performed and malnutrition and inflammation components evaluated. Serum concentrations of Omentin-1 and insulin were measured by using ELISA.

Results: Serum Omentin-1 levels in CKD patients were significantly lower compared to the healthy controls. Further analysis revealed that decrease in omentin in CKD patients is due to the reduced omentin levels in diabetic subgroup. There was a significant difference in serum Omentin-1 levels between non-diabetic (324.2 ± 47.7 ng/mL) and diabetic (189.4 ± 31.2 ng/mL) CKD subgroups ($p < 0.01$). Omentin levels were lower in stage 2 and 3 CKD but not stage 4 CKD patients compared to controls. An increase in inflammation and malnutrition components were correlated with a decrease in the serum level of Omentin. Omentin-1 measurements of the patients according to MI components were shown in Table 1.

Conclusions: Diabetes mellitus and inflammation are responsible from the decrease in serum levels of Omentin in CKD, however, this reduction resolves due to the failure of degradation and excretion of omentin when creatinin clearance falls below 30 mL/dk (stage 4 CKD).

SP152 Table 1: Omentin-1 levels in malnutrition-inflammation (MI) subgroups

Number of MI components	Number of CKD patients (n=64)	Omentin-1 levels (ng/mL)
None	29 (45%)	307.4 ± 172.2
One component	24 (38%)	191.3 ± 84.7^a
Two components	11 (17%)	145.8 ± 67.3^b

One-way ANOVA test results was demonstrated in table 4. The ANOVA test p value was 0.019. CKD: Chronic kidney disease; MI: malnutrition, inflammation.

^a Omentin-1 difference between one MI component versus no MI components, $P = 0.022$.

^b Omentin-1 difference between two MI components versus no MI components, $P = 0.013$; Omentin difference between two MI components versus one MI component, $P = 0.042$.