P-339 EFFECT OF VITAMIN C SUPPLEMENTATION ON SYSTOLIC, DIASTOLIC, PULSE PRESSURE AND LIPIDS: A RANDOMIZED CONTROLLED TRIAL
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This study was designed to test the effect of vitamin C supplementation on systolic, diastolic, pulse pressure and lipids in patients with stage I hypertension. Eligible patients were randomized to 500mg, 1000mg or 2000 mg of vitamin C after a run-in phase of placebo for 4 weeks. During each visit of the follow-up period, which lasted 8 months, a detailed history including medication changes, and physical examination, which included 3 blood pressure readings were performed. A one-week dietary diary was filled prior to each visit. 54 patients were eligible and 31 patients (mean age= 62±2 years, 52% males, 90% whites) were randomized to the three doses of vitamin C. Overall compliance was 48%±2%. Mean systolic blood pressure (SBP) and diastolic blood pressure (DBP) decreased during the vitamin C supplementation phase (mean SBP dropped by 4.5±1.8 mm Hg (p<0.05) and DBP by 2.8±1.2 mm Hg (p=0.05)). This effect was significant for only one month of supplementation but the trend persisted. Pulse pressure (PP) also decreased during the vitamin C phase, but the difference was not statistically significant (mean PP was 53.3±2.4 mm Hg in the placebo phase vs 51.3 mm Hg during the vitamin C phase, p>0.05). There was no difference between the three vitamin C groups (p=0.48). There was no reported intolerance to vitamin C. There was no significant change in serum lipid with vitamin C supplementation, however, there was a trend for a decrease in cholesterol (decrease by 1.5±0.6 mg/dl, p=0.75), Triglyceride (decrease by 2.3±1.48 mg/dl, p=0.87), low density lipoprotein (decrease by 2.1±3.2 mg/dl, p=0.52), and an increase in high density lipoprotein (increase by 2.1±3.2mg/dl, p=0.32) after 6 months of vitamin C supplementation. There was no difference in lipids between the three vitamin C groups. Vitamin C supplementation lowers blood pressure in mildly hypertensive patients. The effect is on SBP and DBP but not PP. Lipids seem to be unaffected by vitamin C supplementation.

Key Words: Vitamin C, Lipids, randomized controlled trials

P-340 INFLUENCE OF LOSARTAN VS ENALAPRIL ON SERUM POTASSIUM IN NORMOTENSIVE PATIENTS WITH DIABETIC NEPHROPATHY AND NORMAL RENAL FUNCTION
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ACE-inhibitors and angiotensin II receptor blockers (ARB) are able to decrease proteinuria in diabetic nephropathy and several glomerular diseases and they can slow the progression of chronic renal failure in some neprhopathies.

However, it has been demonstrated that ACE-inhibitors and ARB increase the serum potassium levels in patients with chronic renal failure or diabetes. Previous results indicate that increases in serum potassium are less likely with Valsartan (ARB) compared with Lisinopril (ACE-inhibitor) in people with renal insufficiency (Bakris GL et al.;Kidney Int 2000;58(5):2084-92).

Objective: This is a comparative study between Enalapril and Losartan on the changes in serum potassium in normotensive patients with diabetic nephropathy and normal renal function.

Design and Methods: The study was an open, crossover design with each period lasting 2 months. 10 patients (5M, 5F) with type 2 diabetes, normotensives, proteinuric and with normal renal function were included in the study.

Results: There was no significant difference in serum potassium between Enalapril vs Losartan periods 5.52±0.55 vs 5.48±0.51 mM/l. There was no difference in blood pressure control, weight, serum creatinine or proteinuria excretion 1.99±0.57 vs 2.09±0.61 gr/24 hours.

Conclusions: Enalapril and Losartan increase serum potassium to similar level in patients with diabetic nephropathy and normal renal function.

Key Words: enalapril, potassium, Losartan

P-341 DIETARY POTASSIUM PREVENTS RENAL DYSFUNCTION AND UPREGULATION OF LOX-1 GENE IN DOCA-SALT RATS
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Objective: To determine the effects of dietary potassium loading on renal function, blood pressure (BP) and oxidative stress in DOCA-salt rats as a model of mineralocorticoid hypertension.

Design and Methods: After unilateral nephrectomy, male 8-week-old Sprague Dawley rats were divided into 3 groups: a) standard chow (0.66% NaCl, 1.34% KCl), b) NaCl-loaded chow (8% NaCl, 1.34% KCl), or c) NaCl and KCl-loaded chow (8% NaCl, 8% KCl). They were weekly subjected to DOCA treatment (100 mg/kg BW). After 2 weeks, BP, plasma and urinary parameters including biomarkers of oxidative stress (plasma 8-epi-PGF2-alpha and 24-hr urinary 8-OH-dG) were determined. Kidneys were subjected to northern blotting to analyze the expression of LOX-1 (lectin-like oxidized LDL receptor-1) gene.

Results: Treatment with DOCA-salt significantly increased systolic BP, associated with renal hypertrophy, renal dysfunction (the increase of s-Cr, proteinuria), and upregulation of LOX-1 gene. Although potassium overdid not have any hypertensive effect, it ameliorated renal dysfunction, renal LOX-1 gene expression, and the increase of markers of oxidative stress in DOCA-salt rats.

Conclusion: These data support the notion that, in mineralocorticoid hypertension, dietary potassium has a protective effect on the renal function independent of BP.

Key Words: dietary potassium, oxidative stress, mineralocorticoid

P-342 REDUCING SODIUM INTAKE REDUCES AMBULATORY BLOOD PRESSURE IN HYPERTENSIVE PATIENTS
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Background: We previously reported in untreated hypertensive patients that 24 hour urinary sodium excretion correlated with diastolic pressure (r=0.52, p= 0.0003) and ambulatory diastolic pressure (r=0.53, p=0.01). We therefore investigated the effect of a diet low in sodium on blood pressure.

Methods: Thirty-six patients (M:F, 18:18; age 45±12 years) with untreated mild essential hypertension were randomised to drug treatment (hydrochlorothiazide 25 mg daily [n=12] or metoprolol 100 mg daily [n=8]) or non-pharmacological treatment (n=16) for 6 months. Ambulatory blood pressure measurements were performed.

Results: There was a significant decrease in ambulatory systolic and diastolic blood pressure in the drug treatment (19±3 mmHg and 11±2 mmHg) and diet group (10±2 mmHg and 6±1 mmHg). In the non-pharmacological group, the decrease in sodium intake correlated with the