

defined as the first hospitalization with a diagnosis of chronic kidney disease.

Results: At baseline, 146 (19%) and 60 (7.6%) patients met metabolic syndrome and microalbuminuria criteria, respectively. After a median follow-up of 11.6 years, renal end-point was reached in 15.8% of patients with metabolic syndrome and in 8.9% of those without it ($P = 0.0087$). The risk of renal events increased progressively starting from patients with neither metabolic syndrome nor microalbuminuria, to patients with only one of these abnormalities, and then to those with both. Significant interaction was observed between metabolic syndrome and microalbuminuria. Patients with concomitant occurrence of metabolic syndrome and microalbuminuria at baseline showed a greater than 5-fold risk of renal outcome as compared to patients with neither of these two risk factors. This risk became even higher when data were adjusted for potential confounders.

Conclusions: Metabolic syndrome and microalbuminuria are independent and interactive predictors of renal outcome in non diabetic patients with primary hypertension.

2D.04 ARE 3 SAMPLES MANDATORY TO CONFIRM A MICROALBUMINURIA?

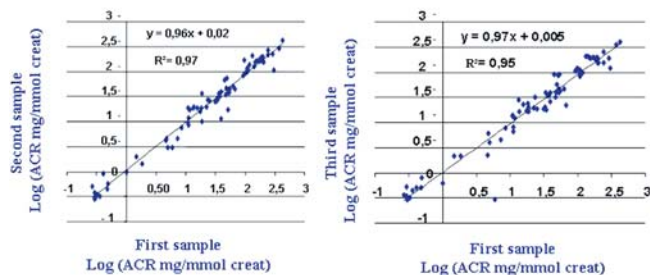
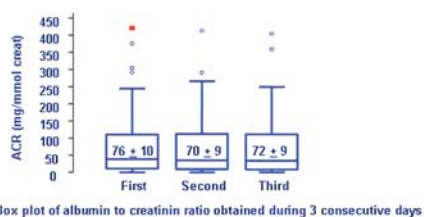
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Objective: Urinary albumin excretion is subject to an intra individual variability. Thus, for research purposes, it is recommended to assay microalbuminuria on three urine samples collected over a short time interval. The objective of our analysis was to check the usefulness to triplicate samples to make sure of the albuminuric status of a diabetic patient.

Methods: Microalbuminuria is a urinary excretion of albumin to creatinin ratio (ACR) between 2.5 and 25 mg/mmol creat in man and between 3.5 and 35 mg/mmol of creatinine in woman. Lower and upper thresholds are usually called normoalbuminuria and macroalbuminuria respectively. We present the not planned retrospective analysis of 82 triplicates (3 consecutive days) morning urine samples obtained during the follow-up of 47 type 2 diabetics (85 % hypertensives) who participated in 3 international studies (ROAD-MAP, AVOID, ALTITUDE). All the measurements (immunoturbidimetry) were performed on fresh urines in the same laboratory. Concordance was obtained if the second and/or the third sample confirmed the albuminuric status obtained from the first sample.

Results: 17 % of the patients were normoalbuminuric, 27 % microalbuminuric and 56 % macroalbuminuric. Mean ACR \pm SEM for the 3 successive samples were 76 ± 10 , 70 ± 9 and 72 ± 9 mg/mmol creat. The concordance rate was 98.8 %. Only a man whose ACR was close to the microalbuminuric upper threshold had 2 clashing values in the successive samples. The coefficient of variation of the immunoturbidimetric method (4 %) widely explains this result.

Conclusion: Our results clearly show that it is useless to repeat albuminuria measurements to define the albuminuric status of hypertensive diabetics subjects.



2D.05 PROXIMAL SODIUM REABSORPTION IS AN INDEPENDENT PREDICTOR OF HYPERTENSION

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Objective: Given the association between salt-sensitivity of blood pressure (BP) and risk of future hypertension (HPT), we investigated the predictive role of alterations in segmental renal tubular sodium handling in the development of HPT during the 8 year follow-up of the Olivetti Heart Study participants.

Methods: A selected sample (n=314) of OHS population was examined at baseline and after 8 years. The participants were included if they were normotensive (SBP/DBP <140/90 mm Hg without anti-hypertensive treatment) and had normal renal function (creatinine clearance - CrCl > 60 ml/min) at baseline. Proximal and distal fractional tubular sodium reabsorption were calculated using the clearance of exogenous lithium.

Results: The baseline sample characteristics were: age 49.3 ± 6.8 yrs, BMI 26.4 ± 2.8 kg/m², SBP/DBP $119.8 \pm 9.6/78.9 \pm 6.4$ mm Hg, CrCl 90.9 ± 18.2 mL/min (M \pm SD). The HPT incidence in 8 years was 52%. The participants who developed HPT (group A) compared with those who did not (group B) had higher baseline SBP (122.4 ± 8.4 mm Hg vs 117.0 ± 10.0 , $p < 0.0001$), DBP (80.6 ± 5.6 VS 77.1 ± 6.7 mm Hg, $p < 0.0001$), BMI (27.0 ± 2.9 vs 25.9 ± 2.6 kg/m², $p < 0.0001$) and fractional proximal reabsorption of sodium (75.8 ± 6.3 vs 73.8 ± 7.3 %, $p = 0.01$). At logistic regression analysis using standardised variables, a 1SD-higher proximal sodium reabsorption at baseline predicted a 44% greater risk of HPT in 8 years (95% C.I. 13–84, $p = 0.003$), independently of baseline SBP (OR: 1.88, 95% C.I. 1.43–2.47, $p < 0.001$), BMI (OR: 1.51, 95% C.I. 1.16–1.95, $p = 0.002$), age and creatinine clearance used as an index of glomerular filtration rate.

Conclusions: In this sample of healthy adult male population, proximal sodium reabsorption indexed to glomerular filtration rate was an independent predictor of future HPT.

2D.06 RENAL AND CARDIAC RENALASE EXPRESSION DURING NORMAL AND HIGH-SALT DIETS IN CHRONIC KIDNEY DISEASE RAT MODEL

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Renalase, a new regulator of cardiac function and blood pressure (BP), is a monoamine oxidase secreted into blood mainly by the kidney but also by the heart and other tissues. A reduced renalase expression was described in patients and in animal models of chronic kidney disease (CKD) and hypertension. In addition, the renalase knockout mouse is hypertensive and exquisitely sensitive to cardiac ischemia. The aim of the present study was to examine the influence of high sodium intake (HS) on renal and cardiac expression of renalase in rats submitted to 3/4 nephrectomy (3/4nx) and Sham surgery (Sham).

Ten days after surgery, 3/4nx and Sham animals received a normal (NS) or a HS diet (40mmol/kg/day) during four days. BP and heart rate (HR) were measured daily using a photoelectric tail-cuff pulse detector. Fourteen days after surgery the animals were sacrificed and left ventricle (LV) and renal cortex (RC) were collected for protein quantification by Western-blotting using the anti-renalase polyclonal primary antibody (1:200, Abcam, UK). In addition, cardiac morphometry and transversal cardiomyocyte diameter (TCD) were also evaluated in all groups.

BP, HR and TCD were significantly increased in 3/4nx rats throughout the study. In addition, HS diet significantly increased BP, HR and TCD in both 3/4nx and Sham rats. During NS diet the dimmer form of renalase was significantly reduced in both RC and LV from 3/4nx rats whereas during HS diet both monomer and dimmer renalase forms were reduced in both RC and LV from 3/4nx rats. Moreover, HS diet significantly decreased both monomer and dimmer renalase isoforms in the LV from 3/4nx rats when compared with NS intake.

Our results show that the reduced renalase expression in the LV from 3/4nx rats is further accentuated during HS intake. It is suggested that this may contribute to the increased sodium-sensitivity of BP and enhanced cardiovascular risk in CKD.