Microalbuminuria in Normotensives with Genetic Risk of Hypertension

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Dear Sir,

Microalbuminuria (μA)^b has been observed in essential hypertension and is usually related to an increased glomerular capillary pressure [1]. It is generally accepted that essential hypertension is in part genetically determined, and, recently, renal functional disturbances have been described in normotensive offspring of hypertensive parents (HP). At rest, a higher renal plasma flow (RPF), and a higher glomerular filtration rate (GFR) [2] or only higher renal vascular resistances (RVR) [3] have been observed in HP compared to normotensives (NT). During a pharmacological challenge with a calcium entry blocker, an increased efferent arteriolar vasodilatation and an abnormality in tubular sodium reabsorption had been observed in HP related to NT [4]. The aim of this study was to assess whether microalbuminuria could precede the development of essential hypertension.

The first group of subjects was composed of 10 normotensives (NT: 5 men and 5 women, mean age 36.1 ± 2.7 years, range 32–41) without any familial history of hypertension. In the second group 10 normotensive subjects were included (HP: 5 men and 5 women, mean age 31.4 ± 7.1 years, range 24–42) who had at least one hypertensive treated parent. The third group was composed of 10 untreated hypertensives (HT: 5 men and 5 women, mean age 42.3 ± 6.7 years, range 32–55). Casual systolic and diastolic blood pressure was similar in NT and in HP (table 1). Diastolic blood pressure in HT ($\text{DBP} = 99.2 \pm 6.6$ mm Hg; evolving for 1.7 ± 3.0 years) was significantly higher than in NT ($\text{DBP} = 67.4 \pm 8.7$ mm Hg) and in HP ($\text{DBP} = 67.6 \pm 4.6$ mm Hg). The fol-

^a $p < 0.05$ vs. NT; ^b $p < 0.05$ vs. day; ^c $p < 0.05$ vs. HP, Mann-Witney test. SBP = Systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; V = urinary flow rate; UD = undetectable: $< 6 \mu\text{g/ml}$.

lowing measurements were performed 15 days after the inclusion day. Subjects were request to collect separately their 'day' and 'night' urines. After emptying their bladders, subjects collected their urine separately from their time of getting up to their time of going to bed (day urines), then from bed time to the time of getting up (night urines). Albumin was measured in the day and night urines, using an immunoturbidimetric method [5]. Creatinine was measured on blood and urine samples by an automated Jaffé method.

Renal function of the three groups was similar regarding their creatinine clearance (Cr), sodium (UNaV) and potassium (UKV) excretion rate during day and night (table 1). Day and night collection periods were equivalent in the 3 groups. In the 3 groups diuresis and sodium excretion rate increased significantly during the night, while the potassium excretion rate decreased significantly. None of the 10 NT exhibited a detectable microalbuminuria, whereas 7 of the 10 HP and 8 of the 10 HT had a detectable microalbuminuria during the day. In all HP microalbuminuria decreased significantly during the night (so that microalbuminuria was only detectable in 6 HP) but in HT microalbuminuria did not decrease significantly (table 1).

The main result of our study was that most of the HP exhibited detectable microalbuminuria whereas their blood pressure was perfectly within normal limits. Significant renal functional abnormalities have been described in HP [2, 3], but to our knowledge, so far, nobody had ever looked into microalbuminuria in these subjects. This microalbuminuria was present during the daytime and decreased significantly during the night, while in established HT the observed microalbuminuria remained at the same level throughout 24 h. The occurrence of microalbuminuria is generally related to renal hemodynamic changes and/or alterations in glomerular wall permeability. Among the early functional abnormalities described in HP, in one study [2] GFR and RPF were found to increase proportionally, without change in filtration fraction, and thus suggesting a fall in RVR. In

another report [3], however, RVR were found to be increased, but no information was given about intracapillary glomerular pressure. In this connection, it is interesting to note that despite the well-known decline in BP during the night, GFR in our patients remained unchanged between day and night, suggesting a nocturnal decrement in RVR, which might account for the fall in microalbuminuria. Prospective studies are needed to determine whether microalbuminuric HP will develop hypertension. If so, determination of the level of microalbuminuria could be useful as a predictive test for hypertension.

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