Predictors of the Evolution of Microalbuminuria

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Albuminuria, either micro- or macro-, is considered as an independent cardiovascular and renal risk factor that must be determined in any patient with diabetes and with arterial hypertension. The presence of microalbuminuria has also been shown to be a risk factor of similar magnitude in the general population, although the possibility of its estimation in the whole population remains as an elusive objective. Recently, 2 relevant issues related to albuminuria have been published: first, prevention of development of microalbuminuria has been demonstrated as an attainable objective in type 2 diabetic patients and, second, diminution of the urinary excretion of albumin has been shown to be followed by a significant decrease in the development of cardiovascular events and death. All of these facts enhance the need to improve our knowledge on predictors of the development and evolution of microalbuminuria. Further progression to macroalbuminuria occurs in a significant number of cases, particularly, but not solely, in diabetic patients, indicating the presence of overt renal disease. Such a progression can be seen in nondiabetic patients with arterial hypertension and also in patients with advanced cardiovascular disease.

The present issue of Hypertension contains a substudy of the Prevention of Renal and Vascular EnD stage Disease (PREVEND) study in which the authors analyze the parameters that determine baseline urinary albumin excretion, as well as the predictors of the response obtained with the administration of an angiotensin-converting enzyme inhibitor during the first 3 months of follow-up in a group of 384 microalbuminuric subjects with apparently normal renal function. As could be expected, baseline values of albuminuria were determined by blood pressure (BP) levels. They also correlated inversely with the level of estimated glomerular filtration rate (GFR) and positively with the urinary sodium excretion. A positive correlation between BP and albuminuria has been amply recognized in multiple studies, and since the first description by Parving et al., BP control is usually accompanied by a significant fall in this parameter. The relationship between renal function, determined as estimated GFR found by the authors, is interesting because, as described in the article, all of the subjects included in the substudy presented with a normal renal function at baseline. The presence of an increased amount of albumin in urine accompanying a normal or increased GFR value can be seen in diabetic patients and less frequently in nondiabetic hypertensive subjects. On the other hand, the prevalence of microalbuminuria and macroalbuminuria has been shown to increase with the progressive decay in GFR observed in hypertensive patients. The finding of the authors that albuminuria correlates with estimated GFR in nondiabetic subjects (mostly normotensive or prehypertensive subjects) further enhances the liaison between cardiovascular risk and renal function since the very early stages of cardiovascular and renal disease. The relationship between natriuresis and albuminuria is also interesting. As recognized by the authors, this finding has been described previously, but the mechanism(s) underlying this finding remain unexplained. An elevated increase in dietary salt and its expression as an elevated natriuresis is particularly harmful in those individuals either normotensive or hypertensive who are salt sensitive. This group of subjects has been shown to present more frequently established cardiovascular disease and also an elevated amount of albumin in urine. Another possible link between salt and microalbuminuria could lie on the frequent presence of metabolic syndrome in the general population. Subjects presenting the characteristics of metabolic syndrome are more frequently microalbuminuric, and accompanying obesity could contribute to explain an increase in salt sensitivity. It should be of interest to analyze whether, in population-based surveys like that included in the PREVEND study, the presence of albuminuria correlated with an increased salt sensitivity that could contribute to explain the relation between albuminuria and natriuresis.

The analysis of the predictors of response to the administration of an angiotensin-converting enzyme inhibitor showed that fosinopril use and BP reduction independently predicted the change in urinary albumin excretion. It was also observed that the higher the baseline level of albuminuria, the higher was the drop in this parameter in response to the suppression of the renin-angiotensin-aldosterone system (RAAS). It has been shown that BP control obtained in the absence of suppression of the RAS can be accompanied by a fall in albuminuria. However, as shown by many studies, it is through the conjunction of an adequate control of BP and an adequate suppression of the RAAS that the most effective decrease in albuminuria is obtained. In agreement with this assertion, the present study shows how the use of fosinopril and the reduction in BP independently predicted the change in urinary albumin excretion. Interestingly, this took place in a group of subjects (predominantly normotensive or prehypertensive) indicating that when accompanied by an elevated global cardiovascular risk, as in microalbuminuric subjects, a drop in BP from values <140/90 mm Hg is accompanied by positive results in a relevant risk factor, such as microalbuminuria. This fact raises the possibility of establishing a new goal BP <120/80 mm Hg in prehypertensive patients with microalbuminuria and that the attainment of that goal must contemplate an adequate suppression of the RAAS. This

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concept should be applicable not only to diabetic patients but also to prediabetic patients in whom an elevated prevalence of microalbuminuria can be found even in the presence of slightly elevated BP levels.12

In summary, as the Figure shows, microalbuminuria is an independent cardiovascular risk factor. It should be early detected to improve global risk stratification and to initiate an adequate intervention to reduce urinary albumin excretion, including an adequate BP control, with suppression of the RAAS and low-salt intake, together with appropriate intervention on other associated cardiovascular risk factors.

Disclosures

None.

References


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