Renal Function in Primary Aldosteronism

To the Editor:

We read the interesting article of Rossi et al.1 investigating the involvement of the kidney in a large group of patients with primary aldosteronism (PA) who were recruited in a multicentric, Italian study. In these patients, the authors observed greater urinary albumin excretion (UAE) and higher prevalence of microalbuminuria than in essential hypertensive (EH) control subjects1 in the presence of comparable glomerular filtration rate (GFR) values, thus supporting the contention that renal damage is more prominent in PA.

However, as the same authors acknowledge in their discussion, the results are somehow deviant from those of previous studies in which glomerular hyperfiltration was consistently shown in PA patients, even using different methods for GFR measurement.2–4 We have recently reported significantly increased GFR in 50 patients with PA (27 adrenal adenoma; 23 idiopathic aldosteronism) in comparison with patients with EH and normotensive subjects who were strictly matched for age, sex, body mass index, and, limited to the hypertensive groups, for severity and duration of hypertension.5 UAE was greater in PA than EH patients, but this difference was eliminated when the urinary albumin:creatinine ratio was considered, suggesting that renal hyperfiltration is a major determinant of increased urinary albumin loss in PA. Therefore, in patients with PA, microalbuminuria might reflect only functional and potentially reversible abnormalities initiated by glomerular hyperfiltration.

This hypothesis has been addressed in our study in which PA and EH patients were prospectively followed for a mean of 6.4 years (range: 3 to 11 years) after adrenalectomy or treatment with mineralocorticoid receptor antagonists. In patients with PA, we observed a significant decline of both GFR and UAE, reaching values comparable with those of patients with EH during the initial 6 months of follow-up. The subsequent rate of decline of GFR was comparable in PA and EH patients, whereas, in the presence of optimal blood pressure control, the progression of albuminuria was irrelevant in both groups. Very similar results were reported in a 6-month follow-up study performed on adrenalectomized patients by Ribstein et al.4 These results suggest that PA is characterized by partially reversible renal dysfunction in which elevated albuminuria is, at least in part, a marker of a dynamic rather than structural renal defect.

The study of Rossi et al.1 is important because, once again, it draws attention to the possible deleterious effects that aldosterone can exert on several target organs independent of its influence on blood pressure. With specific regard to the kidney, however, we should be cautious before dismissing the time-honored notion that PA is relatively “benign” and look for more substantial evidence obtained in prospective long-term studies.

Disclosures

None.

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