Which Antihypertensive Treatment in Renal Vasculitis?

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Dear Sir,

Hyperreninemia along with elevated levels of plasma aldosterone may be an important factor in the pathogenesis of hypertension in patients with renal vasculitis [1].

On the other hand, while the treatment with beta-blockers appears sometimes uneffective in reducing blood pressure [2], the recommended use of angiotensin-converting enzyme (ACE) inhibitors [1] might prove potentially unfavorable in patients whose glomerular filtration rate (GFR) is dependent upon elevated levels of angiotensin II.

A 36-year-old woman was hospitalized because of hemoptysis, headache and arterial hypertension (mean blood pressure 170/110 mm Hg); creatinine clearance was 50 ml/min and proteinuria 1.5-2.0 g/day. Marked hyperreninemia (20 ng/ml/h) and hyperaldosteronism (110 ng/dl) were present (normal values from 0.2 to 2.0 ng/ml/h and 2.0 to 12 ng/dl, respectively). Renal vasculitis was demonstrated by the findings of microaneurysms in the arcuate and interlobular arteries on renal arteriograms and confirmed by histologic examination of renal arterioles.

Orally administered prednisone (1.5 mg/kg body weight) was begun; a treatment with the ACE inhibitor lisinopril (20 mg/day orally) was also started and it significantly lowered blood pressure (mean of 145/85 mm Hg) in both the supine and upright postures. A week later, a reevaluation of renal function showed a marked increase in both blood urea and serum creatinine (210 and 6.1 mg/dl); serum potassium was 6.7 mEq/l while proteinuria decreased to 0.5 g/day. Lisinopril was stopped and amlodipine, a calcium-channel blocker, started (10 mg/day, orally).

Blood pressure levels were effectively controlled, blood urea and serum creatinine decreased to 60 and 2.5 mg/dl, while proteinuria increased to 1.5 g/day. In order to obtain a more satisfactory control of proteinuria, again lisinopril (10 mg/day) was added to amlodipine.

Two weeks later, proteinuria was markedly reduced (0.4 g/day) and blood pressure was effectively controlled; on the other hand, because of a significant worsening of renal function (blood urea 130 mg/dl and serum creatinine 5.5 mg/dl), we had to stop definitively lisinopril administration (table 1). While confirming that the renin-angiotensin system is essential for the maintenance of GFR during a condition of diffuse and severe renal ischemia, our data also clearly show that a significant blood pressure decrease is obtained during treatment with the calcium antagonist amlodipine. Since blood pressure reduction was shown independently of substantial changes in plasma renin activity, it is suggested that additional mechanisms, besides those exerted by angiotensin II, might be responsible for hypertension in our patient.

References

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Table 1. Effect of treatment on renal function and the renin-aldosterone system
Basal Values Lisinopril Amlodipine Amlodipine 10 mg/day plus Lisinopril (20 mg/day) (10 mg/day) (10 mg/day)

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