Diastolic Blood Pressure and Progression of Chronic Renal Failure

K. Kazumasa Shimamatsu, MD, Shimamatsu Naika Iin (Clinic), Futsukaichi 709-1, Chikushino City 818 (Japan)

Dear Sir,

An increase in blood pressure has been considered as one of the factors which may contribute to the progression of chronic renal failure in patients with renal disease [1–5]. However, it has not been discussed in detail which blood pressure, i.e. the systolic or the diastolic (or mean), is more responsible for the progression of renal failure.

Shimamatsu et al. [1] have previously reported that the diastolic blood pressure (but not the systolic) was significantly correlated with the progression (derived from plots of the reciprocal serum creatinine versus time) of renal failure in patients with chronic glomerulonephritis. As well, the progression rate of renal failure in patients with a diastolic blood pressure of less than 90 mm Hg was significantly lower than that in patients with one of more than 90 mm Hg [1]. Bergstrom et al. [2] also reported that the retardation of progression of renal failure was associated with an improvement in diastolic blood pressure control from 93 to 90 mm Hg. On the other hand, Oldrizzi et al. [3] showed that the mean blood pressure did not affect the progression of renal failure.

A recent paper by Brazy et al. [4] confirmed that a diastolic (but not systolic or mean) blood pressure of less than 90 mm Hg was associated with a slower rate of progression to end-stage renal disease.

In this context, the study by Eliahou et al. [5] is of interest; they reported a favorable effect of the calcium channel blocker nisoldipine on the progression of renal failure. These authors stressed the beneficial effect of other factors rather than blood pressure control because no significant difference in the reduction of systolic blood pressure was observed between the nisoldipine-treated group and the group treated with placebo/standard antihypertensives. However, they also described a significant reduction in diastolic blood pressure from 90 to 85 mm Hg (p < 0.03) in the nisoldipine group while no significant change (from 93 to 91 mm Hg) occurred in the placebo group. Although the authors thought that this change in diastolic pressure was too small to be of clinical importance, I think that this significant difference in changes in diastolic pressure should not be ignored in clinical practice from the viewpoint of long-term control of blood pressure.

These clinical studies suggest that the diastolic blood pressure may affect more significantly the progression of renal failure than the systolic or mean blood pressure, and also, from a practical point of view, the control of a diastolic blood pressure of less than 90 mm Hg may be important to preserve the renal function or to blunt the progression of chronic renal failure.

References