Blunted renal responses to angiotensin II infusion in lifetime captopril-treated spontaneously hypertensive rats

蔡世音

Wu JN;Tsai SY and Hsieh WY

摘要

Abstract

Previously, we had found that inhibition of the renin-angiotensin system in the early lifespan of spontaneously hypertensive rat could prevent the development of hypertension in this animal model. In the present study we evaluated the responses of blood pressure and renal function to intracerebroventricular administration of angiotensin II in long-term captopril-treated spontaneously hypertensive rats. Spontaneously hypertensive rats had been mated and their pups were treated with captopril through drinking water after birth. Age-matched Wistar-Kyoto and spontaneously hypertensive rats drinking tap water were used as control groups. At 4 months of age, the basal mean arterial blood pressure of captopril-treated hypertensive rats was the lowest among those of controlled hypertensive and normotensive rats (98+/-5 vs. 160+/-4 and 126+/-4 mmHg, respectively). Intravenous administration of angiotensin II caused similar increments of blood pressure in all rat groups. However, intracerebroventricular administration of angiotensin II to captopril-treated hypertensive rats induced a significantly less increase of arterial blood pressure in comparison with other groups. The sensitivity of baroreflex in captopril-treated hypertensive rats was also the lowest among all rat groups. The basal urine flow, sodium and potassium excretion rates, and osmolar clearance of captopril-treated hypertensive rats were significantly higher than those of controlled hypertensive rats. Intracerebroventricular infusion of angiotensin II caused significant increases in urine flow, electrolytes excretion, osmolar clearance, and free water reabsorption rate of both normotensive and controlled hypertensive rats. However, the same angiotensin II treatment did not change any of the renal excretion indices in captopril-treated hypertensive rats. Our results suggest that lifetime captopril treatment can decrease the activity of the renin-angiotensin system in the brain of hypertensive animals, which caused increases in basal urine flow and excretion of electrolytes and enhanced the sensitivity of baroreflex. It is likely that changes in the renal and baroreflex functions underlie the prevention of hypertension elicited by long-term

captopril treatment.