Acute Effect of Captopril on Serum Lipid Peroxides Level in Hypertensive Patients

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MIZUNO, K., GOTOH, M. and FUKUCHI, S. Acute Effect of Captopril on Serum Lipid Peroxides Level in Hypertensive Patients. Tohoku J. exp. Med., 1984, 143 (1), 127-128 —— Captopril, an orally active angiotensin-converting enzyme inhibitor, was administered to 15 patients with essential hypertension. The serum lipid peroxides level, aldosterone concentration in plasma and blood pressure decreased rapidly after administration, while plasma renin activity was not significantly changed. It is suggested that inhibition of angiotensin-converting enzyme by captopril offers a possible therapeutic approach to the treatment of atherosclerosis complicated with hypertension. —— lipid peroxides; captopril; renin-angiotensin-aldosterone system; hypertension

Previous studies involving patients with mild hypertension have demonstrated a rise in plasma levels of triglycerides with thiazide diuretics alone (Ames and Hill 1976). Also, it has been well known that increases in triglycerides can occur in patients receiving β-adrenoreceptor-blocking drugs such as atenolol, metoprolol or pindolol (England et al. 1978). Although the clinical significance of such a drug-induced rise in triglycerides concentrations remains to be assessed, it may constitute an added risk in view of the association between high lipid concentrations and atherosclerosis. On the other hand, lipid peroxides, produced from unsaturated fatty acid, have been considered factors relating to atherosclerosis since a positive correlation between the extent of atherosclerotic changes and the content of lipid peroxides in human aorta was described by Glavind et al. (1952). Therefore, it seems to be of importance to evaluate the effect of various antihypertensive drugs on lipid peroxides. We investigated the acute effects of captopril, a recently developed specific inhibitor of angiotensin-converting enzyme, on serum lipid peroxides as well as on the renin-angiotensin-aldosterone system and blood pressure in hypertensive patients.

Fifteen inpatients (8 males and 7 females, mean age 41 years) with essential hypertension were studied. None had been treated with drugs before the study. After drawing 0 min blood samples, 50 mg of captopril was given orally. Blood was drawn at 30, 60 and 120 min after the administration for determination of serum lipid peroxides concentration (Yagi 1975). Mean arterial pressure (MAP), plasma renin activity (PRA) and plasma aldosterone concentration (PAC) were also measured as a parameter of captopril’s effect on the converting-enzyme inhibition. Results were presented as mean ± s.d. Statistical analysis was performed with analysis of variance (Wallenstein et al. 1980).

Table 1 depicts our principal data. Serum lipid peroxides were rapidly decreased from

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a control value of 3.25±1.16 to 2.92±0.94 nmol/ml 30 min after the administration. The overall response was significant (p <0.05). MAP as well as PAC were significantly lowered (p <0.001 for MAP; p <0.01 for PAC), while PRA did not significantly change. No significant relationships were observed between changes in serum lipid peroxides levels and in MAP, PRA and PAC at any time after captopril administration.

Although the mechanisms by which serum lipid peroxides are decreased by captopril are not clear, it is suggested from the results that, in addition to the antihypertensive effect, captopril offers a possible therapeutic approach to the treatment of atherosclerosis complicated with hypertension. However, it is necessary to investigate the effect on metabolism of plasma lipid, when used to treat hypertension. The study is now in progress.

References