Angiotensin-Converting Enzyme Inhibitor Trandolapril Does Not Affect C-Reactive Protein Levels in Myocardial Infarction Patients

To the Editor:

Recently, Wang et al. reported a direct effect of C-reactive protein (CRP) on the angiotensin system. Their findings add to the accumulating evidence that CRP is not only an indirect risk marker for cardiovascular disease but also causally contributes to the precipitation of the disease. An association between CRP and the angiotensin system also raises the question of what the effect is of angiotensin-converting enzyme (ACE) inhibitors on CRP, given that the ACE inhibitors are often used after coronary events.

We studied the effect of the ACE inhibitor, trandolapril, on CRP levels in 80 patients who participated in the TRAndolapril Cardiac Evaluation (TRACE) study, randomized to treatment (0.5 mg daily) or placebo. Citrated blood was collected from these patients during hospitalization after a myocardial infarction but before randomization, and after 1, 3, 6, 9, and 12 months of treatment. CRP was measured in the plasma using a high-sensitivity enzyme immunoassay with polyclonal antibodies to human CRP as catching and tagging antibodies (Dako). The effect of treatment on CRP was analyzed using repeated-measures ANOVA on logarithmically transformed CRP levels.

CRP levels were elevated after the myocardial infarction (geometric mean at randomization, 34.1 mg/L [coefficient of variation {CV}, 1.36]) and had returned to normal levels after 1 month, both in the treated group and in the placebo group (2.50 mg/L [CV 1.20] and 3.20 mg/L [CV 1.53], respectively; NS). The levels were not significantly different at any time point during the treatment period.

The results of our study suggest that the association between CRP and the angiotensin system does not include lowering of CRP by ACE inhibitors but may be limited to an effect of CRP on angiotensin I receptor expression as reported by Wang et al.

In conclusion, CRP levels after a myocardial infarction and the beneficial effect of ACE-inhibitor treatment of patients after a myocardial infarction do not seem to include a reduction of levels of the inflammatory marker CRP.

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