Correspondence

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Enhanced Inflammatory Response to Coronary Angioplasty in Patients With Severe Unstable Angina

To the Editor:

We read with interest the recent article by Liuzzo et al1 concerning the enhanced inflammatory response to PTCA in patients with severe unstable angina. They found that PTCA induced an increase in plasma interleukin-6 (IL-6) levels in patients with severe unstable angina, but IL-6 levels did not change after PTCA in stable patients.

We previously reported that plasma IL-6 levels become elevated in patients with acute myocardial infarction2 and that IL-6 mRNA is expressed in human atherosclerotic lesions.3 Therefore, we propose that the main source of plasma IL-6 in coronary artery diseases is the vascular tissue. Recently, we measured the levels of IL-6 and von Willebrand factor (vWF), a marker of endothelial injury, in the coronary sinus blood of patients with stable angina. Blood samples were taken from the coronary sinus immediately before and after and 4 and 24 hours after either PTCA (group A; n=52) or diagnostic coronary angiography (group B; n=7). Levels of IL-6 and vWF in the coronary sinus blood did not change in group B after coronary angiography. Conversely, in group A, the levels of IL-6 and vWF increased significantly 4 hours after the procedure from the baseline value of 3.34±0.56 to 8.20±0.63 pg/mL (P<0.01) and from 113±6.2% to 130±7.3% (P<0.01), respectively, and reached peak values at 24 hours (13.2±1.20 pg/mL and 139±7.4%, both P<0.01 versus baseline). Furthermore, there was a significant positive correlation between IL-6 and vWF levels in the coronary sinus blood at both 4 hours (r=0.38, P<0.01) and 24 hours (r=0.43, P<0.01), suggesting that the mechanism of increased IL-6 after PTCA is related in part to local vascular injury.

Although Liuzzo et al1 found an increase in plasma IL-6 levels after PTCA only in patients with severe unstable angina, we suspect that if blood samples were drawn from the coronary sinus rather than the peripheral vein, more definite conclusions regarding the involvement of inflammatory cytokines in the pathogenesis of acute coronary syndrome could be obtained.

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