Inflammatory Response in Unstable Angina

To the Editor:

I have read with interest the article by Liuzzo et al.1 and their interesting conclusion that “These findings . . . suggest that the magnitude of the acute-phase response is determined to a greater extent by the individual responsiveness than by the type of provocative stimuli.”

I would like to support this theory and to add our experience in this field. Recently, we2 published a study in which 55 patients with coronary artery disease were enrolled. We found that a high level (>100 μg/mL) of an inflammatory protein (serum amyloid type A; SAA) could identify patients with severe unstable angina with a high sensitivity and specificity (r=0.85 and 0.86, respectively). Of the patients studied, 75% experienced an increase in their SAA level 24 hours after angioplasty. An increase of SAA by >100% was associated with an increased risk of restenosis, with a relative risk of 6.4 (P<0.05). We found that there was no correlation between the inflammatory state before angioplasty (or the clinical severity) and the restenosis rate; rather, it was the SAA increase 24 hours after angioplasty that was associated with restenosis. Specifically, the SAA level increased dramatically after angioplasty, unrelated to preangioplasty SAA levels or the clinical state of the patient before the procedure.

I believe that “host traits” (genes? immuno-inflammatory mechanisms?) have an important role in the clinical outcome of every disease process, including coronary artery disease.

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