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

To the Editor:

We congratulate Liuzzo et al on the recent publication of their study, which suggests that disruption of culprit coronary stenoses by PTCA does not necessarily lead to an increase in the level of systemic inflammatory markers (C-reactive protein [CRP], serum amyloid A protein [SAA], and interleukin-6 [IL-6]) in patients with unstable angina. The authors found that serum levels of CRP, SAA, and IL-6 did not change after PTCA in unstable angina patients with normal pre-PTCA (basal) levels but that they significantly increased in those with raised pre-PTCA values. Surprisingly, the authors demonstrated that coronary arteriography also evoked a detectable transient elevation of CRP, SAA, and IL-6 serum levels in unstable angina patients with high basal values. Pre-PTCA and pre–coronary arteriography serum levels of CRP and SAA closely correlated with the corresponding postintervention peak values of both PTCA and coronary arteriography.

As the authors emphasize, it is well documented that elevated levels of acute-phase proteins are associated with an unfavorable short- and long-term prognosis in patients with coronary artery disease; conversely, patients with CRP levels within the normal range tend to have a better prognosis. In light of these data, it would be interesting to know whether the 2 well-matched subgroups of study patients with unstable angina, ie, those with versus those without acute-phase protein response, show the same or different clinical stability and/or therapeutic responsiveness in the pre- and post-PTCA periods. Furthermore, because coronary arteriography could also trigger additional acute-phase protein elevation in a significant proportion of patients with unstable angina, should this transient moderate increase of systemic inflammatory response after coronary arteriography (like PTCA) also be considered a marker for instability of culprit coronary lesions in unstable angina patients with raised basal levels of acute-phase protein?

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Circulation. 1999;100:e95
doi: 10.1161/01.CIR.100.19.e95
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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