Inflammatory Response in Unstable Angina

To the Editor:

We read with great interest the article by Liuzzo et al regarding the systemic inflammatory markers associated with unstable angina. We would like to also emphasize the fact that the increase of inflammatory markers is present not only in the circulation, as shown in their article, but also in the arterial atherosclerotic wall itself. We have previously measured C-reactive protein (CRP) and interleukin-6 (IL-6) levels eluted from human aortic wall with atherosclerosis. High levels of CRP were present in fatty-streak lesions and uncomplicated fibrous plaques. Low levels of CRP were found only in a few normal areas. As measured by ELISA, IL-6 levels were statistically significantly higher in the fibrous plaques than in normal areas. IL-6 was localized by immunohistochemistry as both intracellular and extracellular deposits. Compared with its serum levels, IL-6 was 200-fold higher in the atherosclerotic wall than in serum. The accumulation of CRP and IL-6 in the atherosclerotic wall is a marker for a local inflammatory process.

Our data suggest that both local production and the accumulation of proteins from plasma may contribute to the increased levels observed in atherosclerotic lesions. Serum amyloid A (SAA) is also present in the atherosclerotic wall. Arterial vessel injury is followed by the release of soluble arterial proteins in circulation. Such constant release of IL-6 from vulnerable atherosclerotic plaques in patients with unstable angina may account for the increased serum basal levels seen by Liuzzo et al in their study.

At 6 hours after PTCA, IL-6 levels were twice the basal level (Table 3 in Reference 1). This early and rapid increase suggests that the liberation of IL-6 from the atherosclerotic wall is responsible for the increased levels seen after PTCA in the patients with unstable angina. On the other hand, all measured proteins peaked at 24 hours after PTCA. This also suggests participation of local release in the increased serum level. The high instability of the advanced atherosclerotic lesions, including plaque fissures and ulceration, suggests that this phenomenon is more prominent in patients with unstable angina. The accumulation of CRP, IL-6, and SAA in uncomplicated lesions may be followed by their release from ruptured plaques and may contribute to the increased serum level.

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