C-Reactive Protein and Lesion Morphology in Patients With Acute Myocardial Infarction

To the Editor:

In acute-phase myocardial infarction (AMI), Sano et al. recently showed a C-reactive protein (CRP) concentration in excess of 3 mg/L to be closely correlated with intravascular ultrasound (IVUS) detection of a ruptured atherosclerotic plaque at the culprit lesion site. The authors concluded that such CRP elevation probably reflected inflammation caused by the plaque rupture.

Certain limitations are to be noted, which affect the significance of their study, as follows.

1. CRP was elevated in >90% of patients presenting with AMI preceded by unstable angina. Other authors found CRP elevation in the first 6 hours of AMI, with troponin T levels remaining normal. Now, 40% of the Sano et al. population had preinfarction angina and one third already showed high troponin T levels <3 hours into AMI. These observations suggest that the study population may have been heterogeneous.

2. IVUS diagnosis of ruptured plaque is difficult when a thrombus is involved. During a 1- to 4-week period in which partial thrombosis after AMI is likely to take place, neither we nor other authors detected more than a third of culprit lesion ruptured plaques. Sano et al., on the other hand, report 56%, which may, therefore, include a significant rate of false positives.

In conclusion, the atherothrombotic mechanisms preceding AMI and the diagnostic limitations of IVUS combine to make it uncertain that there is a temporal link between plaque rupture causing AMI and systemic inflammatory syndrome.

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Response

We are very grateful to Dr Rioufol and colleagues for their comments concerning our article.1

Because C-reactive protein (CRP) is mainly generated in the liver after 6 hours of some stimulus, we consecutively enrolled patients in the first 6 hours of acute myocardial infarction (AMI). A third of the patients already had elevated troponin T levels. As we mentioned in our article, myocardial necrosis is one stimulus for CRP generation. Rioufol et al kindly cited a paper that described elevated CRP in the first 6 hours of AMI, with troponin T levels remaining normal.2 These observations supported our conclusion that elevated CRP in AMI is related to lesion morphology but not to myocardial necrosis. In addition, preinfarction angina always means unstable angina.

The idea that intravascular ultrasound (IVUS) cannot evaluate lesion morphology in the acute phase of AMI because of the presence of a large thrombus burden at the culprit site is one of the most common misconceptions regarding preintervention IVUS for AMI. As we mentioned in a previous article,3 lesion morphology at the culprit site changes dramatically over time. For example, low-echoic thrombus is associated with a lesion in the late stages of AMI, and low echoic thrombus may make IVUS assessment difficult. However, an AMI lesion at an earlier stage may contain only bright speckled material that quickly disappears when a guide wire crosses the lesions or adequate IVUS contrast is injected into the culprit lesion. In other words, the earlier the evaluation with IVUS, the better the images. Additionally, as reported in that previous article, we observed plaque rupture in 37.5% of patients within 12 hours of the onset of symptoms using a 3.2F (30-MHz) IVUS catheter; this is a similar rate to that of Rioufol et al, who used IVUS within 1 to 4 weeks after onset of symptoms. However, in our study, we were able to use a 2.9F (40-MHz) IVUS catheter and used IVUS in acute-phase patients. Furthermore, technical advances in the IVUS contrast used have also contributed to improved detection rates for plaque rupture. We are confident, therefore, in insisting that our plaque rupture group does not include false positives, although the non-plaque rupture group might include some plaque rupture patients.

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