Multiple Atherosclerotic Plaque Rupture in Acute Coronary Syndrome

To the Editor:

In their interesting article, Rioufol et al found that patients with acute coronary syndromes frequently had findings on intravascular ultrasound (IVUS) of ruptured plaques in coronary arteries remote from the artery with the culprit lesion. They use this as evidence that there is a period of overall instability throughout the coronary vasculature at the time of the acute coronary syndrome that has brought the patient to angiographic (and IVUS) study. Although in their introduction they quote studies that note a high risk of future events within a year and an increase in the “incidence of coronary atherosclerosis in the months after a coronary accident,” they do not quantify the period of time during which the “pancoronaritis” may persist.

In a study of the natural history of complex (ruptured plaque) and smooth coronary artery lesions performed by analyzing serial coronary arteriograms, we found that complex lesions were frequently preceded by minimally occlusive lesions or seemingly normal vessel segments and that complex lesions were often present in more than 1 vessel simultaneously. However, we also found that in many instances, the complex lesions had been present years before and had not changed in appearance. Moreover, we found that virtually all complex lesions that did not go on to total occlusion (most of the lesions with less than 90% stenosis), which were followed-up with serial arteriograms for a mean of 2.6 ± 1.7 years, did not change in appearance and rarely changed in degree of occlusion. We concluded that first, complex lesions rarely evolve into smooth lesions, ie, that plaque rupture is not the usual mechanism for increase in occlusiveness of smooth-appearing atherosclerotic lesions, and secondly, that the presence of a complex lesion on coronary arteriographic study indicated that plaque rupture had occurred but not necessarily in the recent past; the determination of whether the lesion was acute was more related to clinical and ECG findings.

We would concur that “pancoronaritis” probably does occur, but that the generalized coronary plaque instability may be present for prolonged periods of time (years) or may be episodic, with periods of stability punctuated with repeated short periods of instability. Unfortunately, the coronary arteriographic appearance of a ruptured plaque does not tell how recently the plaque rupture has occurred. Hopefully, this will not also be true of the IVUS appearance of a complex lesion, though only after serial IVUS studies of ruptured plaques are performed will we know with certainty. Hence, until then, the presence of multiple lesions indicative of ruptured plaques does not necessarily imply that all the ruptured plaques had occurred at the time of the culprit lesion, nor does it prove that there is necessarily generalized plaque instability at the time of the patient’s acute coronary syndrome.

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Response

On the basis of repeated angiographic studies showing that complex coronary lesions (CCL) have a certain “chronicity,” Dr Haft raises the possibility that the ruptured plaques associated with culprit lesions may be longstanding, rather than occurring simultaneously with acute coronary syndrome (ACS), which would imply generalized instability. Our intravascular ultrasound-based work was clearly descriptive and it was not possible to date these ruptured plaques associated with culprit lesions; nevertheless, we would like to make a number of remarks concerning these comments:

(1) The retrospective study referred to analyzed CCLs without specifying their clinical context (atherothrombosis or pure chronic ischemia). It is widely accepted, however, that generalized coronary destabilization tends to arise secondarily to ACS and that it is principally in this context that CCLs are liable to show subsequent development.

(2) Other authors have failed to confirm such stability in CCLs. Instead, they report rapid development (8 ± 3 months after the first angiography), suggesting instability associated with this kind of complex plaque.

(3) In our own work, our strict selection of patients during their first event tends to limit the span of prior atherothrombotic development and reasonably implies that “chronic” CCLs, if any, must be of low incidence. To the best of our knowledge, no studies have directly analyzed the incidence of complex plaque in unstable angina, which precludes any comparison with atherothrombosis situations. Even so, from the study by Kaski et al, the incidence of CCL may be estimated at under 30% in their stable angina population, whereas the duration of the development of coronary pathology was not taken into account. By contrast, the incidence of CCL after myocardial infarction or ACS is approximately 40%, indicating a substantial difference from the case of stable angina, and thus arguing for generalized destabilization occurring concomitantly with the acute episode.

In our view, the above facts tend to argue in favor of a simultaneous onset (at least during the first month) of multiple destabilized coronary plaques associated with the ACS culprit lesion. On the other hand, we fully concur with Dr Haft that the duration of post-ACS atherosclerotic destabilization remains to be established, that the developmental tendencies of ruptured plaques is as yet unknown, and that serial IVUS studies are called for.

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Circulation. 2003;107:e65-e66
doi: 10.1161/01.CIR.0000057851.87508.28
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:
http://circ.ahajournals.org/content/107/9/e65

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