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Editorial Comment

Prognostic Implications of Lesion Irregularity on Coronary Angiography*

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The complex coronary plaque. Qualitative analysis of lesion morphology on angiography has generated considerable interest over the past few years (1–3). Significant lesions (<100% occlusion) with irregular borders, overhanging edges or intraluminal filling defects are strongly associated with the presence of the acute coronary syndrome of either unstable angina or acute myocardial infarction. Such "culprit" lesions are found in approximately 70% of patients with either diagnosis, but they are seen infrequently (10% to 20%) in patients with stable angina and no prior history of an acute coronary syndrome.

Although these complex plaques are associated with an acute syndrome, is there any prognostic information that can be derived from their presence? Limited information is available suggesting that lesion irregularity or the presence of filling defects is associated with persistence of symptoms or an adverse in-hospital clinical outcome in unstable angina (4–6). However, because it is customary in unstable angina to intervene with coronary angioplasty or bypass surgery when significant disease is demonstrated, the natural history of these lesions is largely undetermined. An increased incidence of myocardial infarction has also been found on follow-up in patients with irregular lesions independent of the initial clinical presentation (7).

The present study. In this issue of the Journal, Davies et al. (8) report on the effect of lesion irregularity on prognosis in another subset of patients with an acute coronary syndrome. Seventy-two patients undergoing thrombolysis with streptokinase for evolving acute myocardial infarction underwent elective cardiac catheterization 1 to 8 days later. Lesion irregularity as quantified by a plaque ulceration index was related to the subsequent in-hospital clinical course. In the 10 days after angiography, 15 patients with clinical instability had a plaque ulceration index of 6.7 (median value) compared with an index of 3.3 in those with a stable course ($p < 0.001$). Although these data are of potentially great importance, several questions are not answered in the Methods and Results. This missing information, in my opinion, clouds interpretation of their conclusions.

1. Patient selection. Patients underwent catheterization 1 to 8 days after receiving intravenous streptokinase. All patients received intravenous heparin for 4 to 11 days after thrombolysis. This period may have allowed time for remodeling of lesions in those patients undergoing angiography late after infarction, as these same authors (9) reported in a prior study of the same patients. The amount of acute intracoronary thrombus formation is presumably larger in patients with acute myocardial infarction than in most patients with unstable angina (10). Therefore, the remodeling process in patients with complex lesions is likely to be more extensive after infarction than in unstable angina. It would have been preferable to study all patients early and at a set interval after myocardial infarction to study the relation between lesion irregularity and clinical instability. Additionally, we are not told what medical therapy patients received (other than heparin) after thrombolysis. The absence of aspirin in most patients may also have affected the incidence of clinical instability caused by reocclusion as reported in ISIS II (11).

2. Time course of clinical instability. Davies et al. (8) do not tell us when instability occurred after thrombolysis or whether patients with clinical instability underwent angiography earlier after thrombolysis than did patients with a clinically stable course. Furthermore, although we are told that all patients underwent catheterization electively, it is unknown whether any had recurrent pain after thrombolysis but before angiography and whether the 24% of patients with an occluded infarct-related artery on angiography were in any way clinically different from patients without total occlusion. Sixty percent of patients in the clinically stable group required angioplasty or coronary bypass surgery at follow-up. As the timing and indications for revascularization were not given for patients in the stable group it is unknown whether these factors might have changed the relation between lesion irregularity and in-hospital clinical instability. For example, if a large group of patients who were clinically stable were readmitted soon after hospital discharge with recurrent unstable angina or disabling pain, the relation between lesion irregularity and in-hospital instability might be of only marginal clinical significance.

3. Percent diameter stenosis was greater in patients with clinical instability than in patients whose condition remained stable. This finding approached significance ($p = 0.05$) and may have been responsible, at least in part, for the poorer in-hospital course of these patients. Because lesions are often translucent after thrombolysis, a more quantitative approach, utilizing videodensitometry, for example, may have been more appropriate than the method used by Davies et al. (8). Flow characteristics distal to the stenosis were also not reported. Thrombolysis in Myocardial Infarction (TIMI) flow class after thrombolysis has been shown to correlate with subsequent in-hospital survival after myocardial infarc-

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tion. Patients with a TIMI flow class of 3 90 min after thrombolysis had a better survival rate than did patients with lower TIMI flow classes (12,13).

Clinical relevance. These objections represent speculation on my part and should not overshadow the fact that Davies et al. (8) found a strong relation between qualitative morphology and subsequent in-hospital outcome in this group of patients. For those who insist on a quantitative measure of irregularity, this index, as modified by the authors from Wilson et al. (14), can be easily applied and may distinguish patient groups better than subjective indexes of lesion irregularity. Because many lesions classified as complex do not necessarily contain an ulceration, lesions with a high plaque ulceration index require further study to assess their potential for causing continued clinical instability. It has been our experience that these ulcerated lesions are found more commonly after myocardial infarction than after unstable angina. Perhaps these lesions represent true ulceration or fissuring of plaques rather than residual thrombus. Additional studies should be performed with appropriate methodology to assess this finding. If further data corroborate the conclusions of the present study, it would support the use of angiography routinely in the assessment of patients after thrombolysis for acute myocardial infarction.

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