Coronary Arteriographic Lesion of Unstable Angina*

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The morphology of the coronary arteriographic lesions in 109 patients with coronary disease was correlated with their clinical history. Unstable angina, characterized by new onset of angina, angina at rest, or an increase in frequency or ease of precipitation of attacks within the previous two months, was present in 73 patients (group A). The other 36 patients had no history of instability within two months (group B). "Type T" lesions, defined as eccentric narrowing with jagged irregular borders with overlapping or undermined areas, or intraluminal filling defects circumferentially outlined by contrast material were found in 73 percent of group 1 vs 47 percent of group B (p<0.01). The presence of these angiographic lesions suggests that a ruptured atherosclerotic plaque and/or thrombus commonly plays a role in the etiology of unstable angina.

Intracoronary occlusive thrombus with or without rupture of an atherosclerotic plaque has been established as the usual cause of myocardial infarction (MI).1,3 Coronary arteriographic studies during acute MI, experience with the successful use of the fibrinolytic agents, and necropsy studies of patients who died shortly after MI have provided evidence for this central role of thrombosis.5-4 Unstable angina may have a similar etiology, with thrombus forming in a coronary artery on a ruptured plaque, but with less than total or long lasting occlusion of the vessel.5,6 The fact that a large number of these patients proceed to MI and that antithrombotic agents can decrease this high incidence of infarction supports this thesis.7-9 Recently, using meticulous morphologic analysis of the partially occluded segments of coronary arteries in patients who had coronary arteriography soon after the onset of unstable angina, a number of investigators have defined specific arteriographic lesions suggestive of intracoronary thrombus or of complex lesions suggestive of a ruptured plaque with or without associated clot.5,7,10-14 Zack et al10 found 12 percent of patients with unstable angina to have intracoronary filling defects surrounded by dye suggestive of thrombus. Ambrose et al11 found 71 percent of patients with a history of unstable angina to have eccentric lesions with irregular borders in the vessel feeding the area of myocardium that was ischemic on ECG during the acute event. Establishing unequivocally that thrombus plays an important role in unstable angina will open new avenues for therapy and allow the use of fibrinolytic agents earlier in the course of the continuum of the unstable angina—subendocardial infarction—transmural infarction spectrum without waiting for the definitive occurrence of ST elevation that heralds transmural myocardial damage.

To further define the morphology and etiology of the coronary lesion in patients with unstable angina, we have studied the coronary arteriograms of 109 patients and have correlated our findings with the time after the onset of their angina syndrome. We have found that thrombus or a complex lesion suggestive of ruptured plaque and clot is present in 73 percent of patients within two months of the onset of new or unstable angina, whereas only 47 percent have this lesion if their symptoms have been present for longer than two months.

METHODS

A total of 109 consecutive patients with anginal syndromes admitted to St. Michael's Medical Center between September 1984 and November 1985 for coronary arteriography, and were found to have coronary disease, were retrospectively reviewed. The admission histories of these patients were all obtained by the same author, (Haft), and classified (without knowledge of angiographic findings) into the following two subgroups: group A (73 patients) included those individuals whose anginal presentation was unstable in nature. Criteria for instability included (1) new onset of chest pain at rest or at low levels of exertion within two months of arteriography, or (2) a substantial change in a previously stable chronic anginal pattern occurring within two months of study as reflected by an increase in frequency of angina, severity of pain, or pain at rest. Group B (36 patients) included those with stable angina defined as stable exertional chest pain for two months without recent change in character or severity. Patients with prior coronary artery bypass grafting procedures or concomitant significant valvular heart disease were excluded.

Selective coronary arteriograms were performed either by the brachial or femoral routes with multiple views of all vessels obtained in the LAO, RAO, PA, and axial planes and were independently reviewed by two observers blinded to the patients' clinical history. Significant coronary lesions (ie, >50 percent luminal diameter narrowing as seen in orthogonal views) were classified into the following groups according to morphologies outlined by previous

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CHEST / 92 / 4 / OCTOBER, 1987 609
Figure 1. Arteriogram of RCA in the LAO projection with a T lesion characterized by an eccentric narrowing and irregular border in the middle third of the vessel. This lesion is contrasted with the smooth walled non-T lesion in the proximal third of the vessel.

Figure 2. The LAD has a T lesion that is eccentric with an irregular contour after the first diagonal branch. Note that, in contrast, there are two sequential concentric smooth walled lesions, one mild and the other severe in the proximal and middle thirds of the LCF.

Figure 3. A T lesion is seen in a lateral branch of the LCF in the RAO progression. The narrowing is eccentric, its borders are undermined, and its surface appears irregular.

Figure 4. The T lesion in the distal third of the RCA is a filling defect surrounded by contrast material suggesting an intraluminal clot. Investigations\textsuperscript{5,10,11,13} "type T" lesions included those with (1) an area of narrowing with jagged irregular borders with overhanging or undermined edges suggesting ruptured atherosclerotic plaque (similar to Ambrose et al type 2 B lesions), or (2) intraluminal filling defects (with or without staining) circumferentially outlined by contrast material suggesting thrombus (Fig 1 to 5).\textsuperscript{5,10,11,13} Other lesions, not indicative of ruptured plaque or thrombus, included those with smooth concentric or eccentric narowings, total occlusions, or multiple irregularities (not exhibiting characteristics of T lesions) within a single coronary artery. All vessels and all lesions were analyzed. Any diagnostic disagreements were settled by discussion among all three authors and a consensus was reached on each lesion. Comparison between the groups was tested for significance using Student's t-test or chi-square analysis.

Results

The mean age and the sex ratio of patients in group A (with unstable angina) was not significantly different from those in group B (Table 1). The incidence of single, two, and three vessel disease was also similar in the two groups. Among the patients in group A, there was a significantly higher incidence of T lesions, compared to group B (53 patients with T lesions of 73 compared to...
Table 1—Clinical and Angiographic Findings

<table>
<thead>
<tr>
<th>Lesion Location</th>
<th>Group A</th>
<th>Group B</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAD</td>
<td>21</td>
<td>8</td>
</tr>
<tr>
<td>RCA</td>
<td>14</td>
<td>2</td>
</tr>
<tr>
<td>LCF</td>
<td>9</td>
<td>2</td>
</tr>
<tr>
<td>Left main</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2 Lesions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAD &amp; LCF</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>LAD &amp; RCA</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>LCF &amp; RCA</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>LM &amp; RCA</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>LM &amp; LAD</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

*Number of patients with T lesions in parentheses.
†TO, total occlusions.

17 of 36 patients (p<0.01). Of the patients with single vessel disease, nine of 17 in group A had T lesions. An additional five patients had a totally occluded vessel, that would obviate determining if a T lesion had been the initial event. In group B, three of nine had T lesions with two totally occluded. Thus, nine of 12 with analyzable single vessel disease had T lesions compared to three of seven in group B. The numbers are too small to achieve statistical significance. A total of 46 patients in group A had single T lesions and seven had T lesions in two vessels. This was not significantly different from the distribution in group B (15 single T lesions, two double T lesions). The distribution of individual vessels that contained T-lesions also were not different between the groups, although there was a trend toward a higher proportion in group A patients having RCA lesions and a trend toward more in group B having left main lesions. In both groups, the LAD was the vessel most often containing a T lesion. Six of the patients with T lesions had filling defects surrounded by contrast material. All were in group A (8.2 percent of group A).

The relation of T lesions to time after onset of symptoms is presented in Table 2. All of the T lesions occurred in the patients within <six months of symptoms, with most T lesions seen in those less than two months after onset of symptoms.

Table 2—Number of Patients with T Lesions in Relation to Time After Onset of Symptoms

<table>
<thead>
<tr>
<th>Time</th>
<th>0-1 week</th>
<th>1-2 weeks</th>
<th>2-4 weeks</th>
<th>1-2 months</th>
<th>2-4 months</th>
<th>4-6 months</th>
<th>&gt;6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>T lesion</td>
<td>14</td>
<td>8 (2*)</td>
<td>17 (3*)</td>
<td>14 (1*)</td>
<td>14</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Other</td>
<td>12 (5***)</td>
<td>4 (1†)</td>
<td>3 (1†)</td>
<td>1 (1†)</td>
<td>9 (2†)</td>
<td>2 (1†)</td>
<td>8</td>
</tr>
</tbody>
</table>

*Clot.
†Total occlusion.

Discussion

Until recently, evidence for the mechanism of the etiology of unstable angina has been elusive. It had been postulated that unstable angina was due to the transient occlusion of a coronary artery either by a platelet clot or by a transient spasm superimposed on a preexisting area of narrowing or by a combination of both. Coronary arteriographic data analyzed and quantitated in the usual manner have not shown any significant differences in the number of vessels occluded or in the severity of the occlusions between patients with stable and unstable angina. Characterization of the specific morphology of lesions has been more productive, however; Zack et al. and others have described a specific coronary lesion that has a central filling defect surrounded by dye that suggests an intracoronary clot. Post-mortem studies of patients with these lesions have verified that recent thrombus can cause such an arteriographic phenomenon. This morphologic pattern is found in 6 to 45 percent of patients with unstable angina but only very rarely in patients with stable angina. Ambrose et al. have defined another morphologic lesion that is more consistently found in patients with unstable angina. They found complex eccentric lesions with underlined borders in a large percentage of patients with unstable angina. Patients with stable angina had this finding significantly less frequently. These same authors postulated that the lesion represented a ruptured plaque with or without superimposed thrombus. Of interest is the finding that patients who had had successful fibrinolysis with intracoronary streptokinase had a similar lesion in the vessel that had been opened. This observation supports the concept that unstable angina is part of the spectrum of occlusive coronary disease that culminates in transmural MI. Recently, Sherman et al. using angiography, found clot and ruptured plaque to be present in patients who had arteriographic lesions similar to those described by Ambrose et al. In this study, we have investigated the incidence of lesions similar to those described by Ambrose et al and of lesions similar to the filling defects cited by Zack et al in patients who have come to arteriography. The incidence of both of the lesions was significantly higher among those with a definite history of unstable angina within two months of study than in the patients whose angina history showed no recent change. Our findings...
suggest that these lesions are the hallmarks of unstable angina and further suggest that thrombus formation is as important in the etiology of unstable angina as in myocardial infarction.

The presence of a T lesion in a number of patients with historically more stable angina suggests that possibly the lesion can persist for as long as six months without going on to infarction. Alternatively, it is possible that patients without a definitive history of an increase in symptoms may have had a subtle change in their clinical course that had precipitated their visiting their physicians and their subsequent referral for angiography. The failure to find typical T lesions in the 27 percent of patients with unstable angina who did not have them may have been due to the fact that many of the patients in group A had total occlusion of one or more vessels. It is usually impossible to diagnose a T lesion in a totally occluded vessel. It is possible that reendothelialization of defects or clots, or endogenous fibrinolysis of the thrombus parts of these lesions might have altered the appearance of a lesion so that diagnosis of a T lesion would be masked.

Recognition of these lesions that are frequently associated with unstable angina may be of help in deciding management, whether with anticoagulation, aspirin, angioplasty, or coronary bypass. The value of fibrinolytic agents in patients with these lesions remains to be determined.

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REFERENCES