Angiographic-Pathologic Correlations After Elective Percutaneous Transluminal Coronary Angioplasty

Takahiko Naruko, MD; Makiko Ueda, MD; Anton E. Becker, MD; Osamu Tojo, MD; Masakazu Teragaki, MD; Kazuhide Takeuchi, MD; Tadanao Takeda, MD

Background. The local effect of coronary angioplasty is evaluated on the basis of postangioplasty angiograms. Smooth-walled dilation is considered to represent minimal or no injury, whereas intraluminal haziness corresponds with wall laceration. This study correlates the preangioplasty and postangioplasty angiograms with the histopathology of the target sites.

Methods and Results. The study includes 12 patients, each undergoing an elective procedure, and covers 19 angioplasty sites. Smooth-walled dilation and intraluminal haziness were not mutually exclusive. The angiograms were interpreted as smooth-walled dilation (n=3), smooth-walled dilation with intraluminal haziness (n=4), intraluminal and extraluminal haziness (n=5), extraluminal dissection (n=5), spiral-type dissection (n=1), and aneurysm (n=1). The histology of the arterial segments revealed wall laceration in all. Smooth-walled dilation without intraluminal haziness correlated with laceration limited to the intima in two, but with medial injury in one. Smooth-walled dilation with intraluminal haziness correlated with laceration limited to the intima in two and with medial injury in two. Intraluminal and extraluminal haziness corresponded with extensive laceration with deep involvement of the media in each. Extraluminal dissection correlated with a dissection along the shoulder area of the plaque, creating a broad-based flap. The spiral-type dissection corresponded with a true dissection into the plaque-free media. The aneurysm correlated with partial washout of an atherosclerotic plaque.

Conclusions. The angiographic image of intraluminal and extraluminal haziness indicates extensive medial laceration. Smooth-walled dilation, with or without intraluminal haziness, is not a reliable indicator. The study emphasizes the need to reconsider the interpretations of postangioplasty coronary angiograms. (Circulation. 1993;88[part 1]:1558-1568.)

Key Words • percutaneous transluminal coronary angioplasty • restenosis • dissection

The angiographic images taken after initial successful coronary angioplasty are varied and have been classified by Holmes and coworkers1 as smooth-walled dilation, intraluminal haziness, intimal flap or intraluminal split and dissection, and aneurysmal dilation. More recently, intravascular ultrasound has been used to assess the morphology after percutaneous transluminal coronary angioplasty.2,3 These studies reveal major discrepancies with angiographic evaluation, but pathologic correlations have not been documented. To the best of our knowledge, Waller4 is the only one thus far who described the correlates between the angiographic images and the anatomy of the vessel wall at the site of angioplasty in humans. He suggested that a dilated arterial segment with a smooth outerlining correlated with minimal/superficial or no wall laceration, whereas intraluminal haziness, characterized by a "ground glass" appearance of the contrast medium, was indicative of extensive and much more serious injury. These are important connotations with immediate clinical relevance. The present study has been undertaken because of this aspect as well as the fact that the author did not provide pictures showing the morphology in direct relation with the angiographic images. The present study documents angiographic-morphologic correlates in 12 patients, covering 19 sites of an initially successful coronary angioplasty.

Methods

The study is based on 12 patients who had undergone elective coronary angioplasty (9 because of stable angina and 3 because of unstable angina pectoris). The relevant clinical data are summarized in Table 1. The interval between angioplasty and death ranged from 9
TABLE 1. Relevant Clinical Data of 12 Patients Who Underwent an Elective Coronary Angioplasty and in Whom an Angiographic-Pathologic Correlation Was Made

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, y</th>
<th>Sex</th>
<th>Reason for PTCA</th>
<th>Dilated Site</th>
<th>Interval PTCA/Death</th>
<th>Pre</th>
<th>Post*</th>
<th>PTCA Artery Narrowing (%DR)</th>
<th>Maximal Balloon Size</th>
<th>Balloon/Artery Ratio</th>
<th>Cause of Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>57</td>
<td>M</td>
<td>SAP, OMI</td>
<td>LAD (6)</td>
<td>9 h</td>
<td>75</td>
<td>25</td>
<td>8.0 3.5 1.3</td>
<td></td>
<td></td>
<td>Acute occlusion</td>
</tr>
<tr>
<td>2</td>
<td>61</td>
<td>M</td>
<td>SAP, OMI</td>
<td>LAD (6)</td>
<td>2 d</td>
<td>75</td>
<td>0</td>
<td>4.0 3.0 1.2</td>
<td></td>
<td></td>
<td>Sudden death caused by pulmonary embolism</td>
</tr>
<tr>
<td>3</td>
<td>85</td>
<td>M</td>
<td>UAP</td>
<td>LMT (5)</td>
<td>2 d</td>
<td>75</td>
<td>0</td>
<td>8.0 3.0 1.1</td>
<td></td>
<td></td>
<td>Intestinal infarction caused by thromboembolism</td>
</tr>
<tr>
<td>4</td>
<td>55</td>
<td>M</td>
<td>UAP</td>
<td>LAD (6)</td>
<td>1 mo</td>
<td>90</td>
<td>25</td>
<td>12.0 2.5 1.0</td>
<td></td>
<td></td>
<td>Acute myocardial infarction</td>
</tr>
<tr>
<td>5</td>
<td>67</td>
<td>F</td>
<td>UAP</td>
<td>RCA (1)</td>
<td>1 mo</td>
<td>75</td>
<td>0</td>
<td>8.0 3.0 1.3</td>
<td></td>
<td></td>
<td>Intestinal infarction and renal failure caused by cholesterol embolism</td>
</tr>
<tr>
<td>6</td>
<td>62</td>
<td>F</td>
<td>SAP, OMI</td>
<td>LAD (7)</td>
<td>2 mo</td>
<td>75</td>
<td>25</td>
<td>8.0 2.5 1.3</td>
<td></td>
<td></td>
<td>Sudden cardiac death caused by urosepsis</td>
</tr>
<tr>
<td>7</td>
<td>50</td>
<td>M</td>
<td>SAP</td>
<td>LMT (5)</td>
<td>4 mo</td>
<td>90</td>
<td>0</td>
<td>7.0 2.5 1.0</td>
<td></td>
<td></td>
<td>Acute myocardial infarction</td>
</tr>
<tr>
<td>8</td>
<td>67</td>
<td>M</td>
<td>SAP</td>
<td>LAD (6)</td>
<td>4 mo</td>
<td>75</td>
<td>0</td>
<td>4.1 3.0 1.3</td>
<td></td>
<td></td>
<td>Sudden death after PTCA of RCA</td>
</tr>
<tr>
<td>9</td>
<td>58</td>
<td>M</td>
<td>SAP</td>
<td>LAD (7)</td>
<td>7 mo</td>
<td>90</td>
<td>50</td>
<td>6.0 2.5 1.0</td>
<td></td>
<td></td>
<td>Esophageal cancer</td>
</tr>
<tr>
<td>10</td>
<td>73</td>
<td>M</td>
<td>SAP</td>
<td>LCx (13)</td>
<td>8 mo</td>
<td>90</td>
<td>0</td>
<td>10.0 2.0 1.0</td>
<td></td>
<td></td>
<td>Leiomyosarcoma of stomach</td>
</tr>
<tr>
<td>11</td>
<td>73</td>
<td>M</td>
<td>SAP</td>
<td>LAD (6)</td>
<td>1 y 1 mo</td>
<td>90</td>
<td>0</td>
<td>NI 3.5 1.2</td>
<td></td>
<td></td>
<td>Lung cancer</td>
</tr>
<tr>
<td>12</td>
<td>48</td>
<td>M</td>
<td>SAP</td>
<td>RCA (1)</td>
<td>3 y 3 mo</td>
<td>75</td>
<td>25</td>
<td>NI 3.0 0.9</td>
<td></td>
<td></td>
<td>Rupture of aortic dissection</td>
</tr>
</tbody>
</table>

PTCA indicates percutaneous transluminal coronary angioplasty; DR, diameter reduction; SAP, stable angina pectoris; OMI, old myocardial infarction; UAP, unstable angina pectoris; LAD, left anterior descending coronary artery; LMT, left main artery; RCA, right coronary artery; LCx, left circumflex artery; Diag, diagonal; and NI, no information.

* Segment(s) of the coronary arteries that were dilated are shown in parentheses (according to the American Heart Association Committee Report. Circulation. 1975;51[suppl]:5-40.).

** In case of haziness, the outer border was taken as reference.

PTCA indicates percutaneous transluminal coronary angioplasty.

TABLE 2. Angiographic-Pathologic Correlates at 19 PTCA Sites

<table>
<thead>
<tr>
<th>Angiographic Images at Sites of PTCA</th>
<th>Number of PTCA Sites</th>
<th>Pathologic Findings at Site of PTCA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Intimal Findings</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Eccentric</td>
</tr>
<tr>
<td>Smooth-walled dilation</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Smooth-walled dilation with intraluminal haziness</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Intraluminal and extraluminal haziness</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Dissection</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Aneurysmal dilation</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

PTCA indicates percutaneous transluminal coronary angioplasty.
Fig 1. Correlation of preangioplasty (A) and postangioplasty (B) angiograms in a case diagnosed as smooth-walled dilation (patient 4), with the pathology at the target site (C). The target site is indicated by arrows (A, B). The pathologic correlate (C), 1 month after percutaneous transluminal coronary angioplasty, shows that a plaque fissure (arrow) occurred at the border zone between the atherosclerotic plaque (AS) and the adjacent wall (elastic tissue stain, original magnification ×21).

Fig 2. Correlation of preangioplasty (A) and postangioplasty (B) angiograms in a case diagnosed as smooth-walled dilation with intraluminal haziness (patient 12), with the pathology at the target site (C). The target site is indicated by arrows (A, B). The pathologic correlate (C), 3 years and 3 months after percutaneous transluminal coronary angioplasty, shows that the procedure caused superficial injury to the media (M) of the plaque-free wall (arrow), with a localized fibrocellular (F) tissue response (elastic tissue stain, original magnification ×17).
FIG 3. Correlation of preangioplasty (A) and postangioplasty (B) angiograms in a case of intraluminal and extraluminal haziness (patient 6), with the pathology at the target site (C). The target site is indicated by arrows (A, B). The pathologic correlate (C), 2 months after percutaneous transluminal coronary angioplasty, shows that the procedure caused extensive laceration at the border zone between the atherosclerotic plaque (AS) and the adjacent wall (arrow), extending deep into the media (M) and almost leading to rupture. The contours of the initial damage can be traced easily, although the tear is filled largely with fibrocellular (F) tissue (elastic tissue stain, original magnification ×21).

FIG 4. Correlation of preangioplasty (A) and postangioplasty (B) angiograms in a case diagnosed as intraluminal and extraluminal haziness (patient 8), with the pathology at the target site (C). The target site is indicated by arrows (A, B). The pathologic correlate (C), 4 months after percutaneous transluminal coronary angioplasty, shows that laceration occurred at the border zones between the plaque (AS) and plaque-free wall (arrows), extending into the media (M). The sites of laceration are covered by fibrocellular (F) tissue, also extending over the plaque (elastic tissue stain, original magnification ×27).
hours to 39 months. In each case, the preangioplasty and postangioplasty angiograms were available for examination. In these 12 patients, there were 19 sites that were dilated by the balloon, 2 of which occurred at neighboring arterial segments not considered as containing the target lesions (see Table 1, patients 7 and 9). The angioplasty procedure was considered successful when improvement of the luminal diameter was >30%. Maximum inflation pressure at the angioplasty procedure varied from 4 to 12 atm.

Two patients (patients 4 and 7) developed clinical and angiographic evidence of restenosis (defined as a loss of ≥50% of the initial gain) and underwent coronary artery bypass grafting. Follow-up angiographic examinations were performed in 3 other patients. In patients 10 and 12 (Table 1), the follow-up angiographic studies were performed 5 and 28 months after angioplasty, respectively. In these patients there was no angiographic evidence of restenosis. In patient 11, the follow-up angiographic study was performed 11 months after angioplasty. No evidence for restenosis was found in the left anterior descending coronary artery (LAD) (segment 6), but segment 7 of this artery did show restenosis (loss of 75% of the initial gain).

**Angiographic Classification**

The preangioplasty and postangioplasty angiograms were studied independently by three experienced angiographers who were not informed about the clinical and pathologic data. The angiographic changes in the postangioplasty angiogram were classified according to the criteria set by Holmes and coworkers. When all three angiographers were unanimous in their classification, the case was considered typical. In case of differences of opinion, the angiographic change was considered in-between. Such differences occurred only with
respect to the differentiation between smooth-walled dilation and haziness.

In each of the 12 patients, the heart was available for study. The precise site of angioplasty was decided by measuring the distance from the angiograms, using coronary ostia and bifurcation sites as points of reference. The arterial segments involved were sectioned serially at 1-mm intervals, and the blocks were routinely processed. Each 1-mm coronary arterial segment was histologically serially sectioned at 5-μm thickness, and every 10th and 11th section was mounted and stained with hematoxylin-eosin and elastica van Gieson stains, respectively. The histological study was performed by two pathologists who at the time of examination were unaware of the postangioplasty angiographic images. Once the angiographic and the pathologic studies were completed, a correlation was made between the findings.

Results

Angiographic Classification

Following the criteria set by Holmes and coworkers,1 major problems arose with respect to the proper distinction between smooth-walled dilation and those changes designated as intraluminal haziness. In fact, the intraluminal contrast frequently showed haziness (ground glass appearance) irrespective of a smooth or an irregular outer lining of the artery. The magnitude of the problem is best illustrated by the fact that smooth-walled dilation without intraluminal haziness was diagnosed by all three angiographers in only three instances. In 9 cases, the dilemma mentioned above led to disagreement with respect to a unanimous classification of a dilated segment as either smooth-walled dilation or intraluminal haziness and, hence, a categorization as in-between.

For these reasons, the classification of Holmes and coworkers1 was adjusted. Smooth-walled dilation was redefined as being characterized by a smooth outer lining of the arterial segment, present at both sides and without intraluminal haziness. A second category consisted of a smooth-walled dilation but with intraluminal haziness. The third category was characterized by haziness that also affected the outerlining of the segment involved, thus causing a fuzzy outerlining on one or both sides, a condition more properly designated as intraluminal and extraluminal haziness.

Smooth-walled dilation was seen in 3 instances (Fig 1). Smooth-walled dilation accompanied by intraluminal haziness occurred in 4 cases (Fig 2), and intraluminal and extraluminal haziness was diagnosed in 5 cases (Figs 3 through 5). A dissection was diagnosed in 6 cases (Fig 6), and an aneurysmal dilation (Fig 7) was identified in one instance; in neither of these instances was there any disagreement between the three angiographers. An intimal tear or flap (as defined by Black and associates6) and thrombosis were not identified from the angiographic images.

Pathologic Findings

Each segment in which the angioplasty procedure had been performed showed laceration of the wall at microscopic examination, although to a varying extent. Free-

![Image](https://example.com/image.png)
Dilation (C), washout (arrow) of a preexistent atheroma, at this stage with reparative tissue (asterisk) covering plaque hemorrhage (H) (elastic tissue stain, original magnification ×17).

Persistent plaque morphology was categorized as either concentric (occupying the total circumference of the artery) or eccentric (part of the circumference is plaque free). Balloon-induced wall injury was limited to the intimal lesion at 5 sites, whereas 14 sites showed laceration involving the media as well. In 4 of the 5 instances with wall laceration limited to the intimal lesion, the plaque morphology was identified as concentric. In case of injury extending into the media, 13 of the 14 segments involved showed an eccentric lesion. The morphology of the initial lesion, induced by the angioplasty procedure at the time of the intervention, was identified in the vast majority of cases on the basis of the fibrocellular tissue response. In 3 patients (patients 1, 2, and 3) the interval between the angioplasty procedure and death was short (9 hours to 2 days), thus allowing direct identification of the injury evoked by the procedure.

**Angiographic-Pathologic Correlations**

The results are summarized in Table 2. Two of the 3 cases diagnosed as smooth-walled dilation showed a concentric atheromatous plaque with injury limited to the intima (Fig 1). The third case presented an eccentric lesion with a laceration that had extended into the superficial zone of the media.

The 4 cases with an angiographic image of smooth-walled dilation with intraluminal haziness showed a similar histopathological correlation. In 2 cases, the injury was limited to the intimal lesion, both showing a concentric type atherosclerotic plaque. In 2 other cases (1 eccentric and 1 concentric lesion), laceration had extended into the media, albeit rather superficially (Fig 2).

These observations contrast distinctly with the pathology encountered in the 5 cases in which intraluminal and extraluminal haziness was diagnosed. In each of these 5 arterial segments, an eccentric lesion was found, with extensive laceration involving the media (Figs 3 through 5). In 1 patient (patient 9), the catheter had been guided into the LAD (segment 7, the target lesion) and further into the diagonal branch (segment 10, a nontarget site). Balloon dilation led to an intimal laceration at the site of the target lesion but caused extensive injury to an eccentric plaque in the diagonal branch, which resulted in medial dissection (Fig 8).

Six cases (including the 1 nontarget site mentioned above) showed the angiographic image of a dissection, 5 of which showed the extraluminal type as defined by Black and coworkers. In each of these 5 cases, a preexistent eccentric atherosclerotic plaque was present with extensive wall laceration. The injury started at the shoulder region of the plaque and separated the plaque from the underlying preexistent arterial wall spreading in longitudinal direction and basically creating a broad-based flap (Fig 6). The remaining sixth case showed a spiral tear characterized by a double lumen with an almost central slitlike radiolucent area extending in a longitudinal direction (Fig 9). In this instance, a tear had started also at the shoulder region of an atherosclerotic plaque. However, a true dissection occurred that extended into the plaque-free wall, thus producing a false channel within the media. Serial histological sections, moreover, revealed that the false channel had entry sites both at its proximal and distal ends (Fig 9).

The one case with an aneurysmal dilation showed a partial washout of a preexistent atheroma (Fig 7).
FIG 8. Correlation of preangioplasty (A) and postangioplasty (B) angiograms, with the pathology at the site of the target lesion (C) and at a nontarget site (D) in patient 9. The target lesion is indicated by single arrows (A, B); the nontarget site is indicated by double arrows (A, B). At the target site, the postangioplasty angiogram (B, single arrow) shows smooth-walled dilation with intraluminal haziness and wall laceration (C, arrow) limited to the intimal lesion, at this stage (7 months after percutaneous transluminal coronary angioplasty) covered by a small rim of fibrocellular (F) tissue. At the nontarget site, the postangioplasty angiogram (B, double arrows) was diagnosed as a dissection of the extraluminal type; the pathologic correlate (D), 7 months after the procedure, shows that extensive laceration occurred at the border area between the plaque and the plaque-free wall, producing medial dissection, presently largely filled by fibrocellular (F) tissue (elastic tissue stain; C, original magnification ×19; D, original magnification ×30).

Discussion

This study has correlated the angiographic appearances of 19 coronary arterial segments, dilated during an elective angioplasty procedure, with the pathology at the target site. In 3 patients (5 sites), the interval between angioplasty and death allowed direct visualization of the laceration induced. Several important points emerge from this study. In 9 patients (14 sites), the lesions induced were traced on the basis of the fibrocellular tissue response triggered by the injury. In all instances, including the 2 patients who died more than 1 year after the angioplasty (patients 11 and 12), the reparative tissue was clearly identified and distinct from preexistent tissues, such as the atherosclerotic lesion, thus allowing the reconstruction of the initial injury irrespective of possible additional changes that could have altered the plaque. It is pertinent to this study, therefore, that the actual site of the angioplasty always could be traced because of a distinct laceration. Hence, there is no need to consider hypothetical remodeling mechanisms such as "chronic elastic recoil," proposed to explain restenosis without evidence of arterial wall injury.

The angiographic classification proposed by Holmes and coworkers caused problems, in particular because intraluminal haziness, characterized by a ground glass appearance of the contrast medium, and smooth-walled dilation were not mutually exclusive. It is for these
FIG 9. Correlation of preangioplasty (A) and postangioplasty (B) angiograms, with the pathology at the target site in a case diagnosed as a spiral-type dissection (patient 5). The target site is indicated by arrows (A, B). The pathologic correlates reveal that the procedure caused extensive longitudinal dissection. The proximal site of entry (C) occurred at the border zone between the atherosclerotic plaque and the plaque-free wall, with the tear extending into the media (arrow). The dissection led to a false channel (D, asterisk) within the media of the plaque-free wall. At a more distal site (E), the dissection reentered into the original lumen (arrow). L indicates original lumen (elastic tissue stains; C and D, original magnification ×14; E, original magnification ×16).
reasons that we have categorized the angiographic images as smooth-walled dilation without a ground glass appearance of the intraluminal contrast, smooth-walled dilation with intraluminal haziness, and intraluminal and extraluminal haziness. In the latter condition, the outer lining of the arterial segment is fuzzy and irregular and merges with the intraluminal ground glass appearance. A recent study in which haziness was defined as a small radiolucent area with the passage of contrast material (type A dissection, according to Dorros et al) also disclosed confusion regarding the definition of haziness amid other parameters of angiographic judgment.

The concern expressed with respect to the classification of postangioplasty angiographic appearances is important. Waller suggested that once a dilated arterial segment appears as smooth-walled, there is either no underlying injury or the damage is limited to the superficial intima. Holmes and associates also consider compression of a soft atheroma or stretching of a fibrous plaque as the underlying anatomic substrate in such cases. On the other hand, intraluminal haziness has been suggested to be caused by dispersion of contrast medium into fissures within the dilated segment, thus indicative of a much more serious wall laceration.

The present study puts these statements into a wider perspective. Indeed, based on angiography, none of the segments classified as smooth-walled presented extensive wall laceration. Nevertheless, 3 of 7 segments so categorized showed injury extending from the intimal lesion into the media, albeit superficially. Two of these 3, moreover, presented a ground glass appearance of the contrast material. In a given patient, therefore, a smooth-walled angiographic image is not necessarily a good predictor for the underlying pathology. The uncertainties with respect to the underlying pathology are nonexistent, at least in this study, once the postangioplasty angiogram shows intraluminal and extraluminal haziness. Each of the 5 segments with this angiographic label attached to it showed extensive laceration with deep involvement of the preexistent media. It is of additional interest that in each instance the atherosclerotic lesion was eccentric in nature. It is our belief, therefore, that the presently proposed refinements of the classification of Holmes and coworkers have clinical relevance in being more accurate as to the underlying pathologic substrate. Further studies are necessary to evaluate whether or not they also provide useful prognostic clinical information.

Another point related to the above is that none of the cases was devoid of any injury to preexistent tissues. This contrasts with the observations made by Waller, who had 8 angioplasty sites among a series of 76 cases with an angiographic image of smooth-walled dilation.

The pathologic correlate in the case showing an aneurysm on the postangioplasty angiogram is of special interest. The correlation with the pathology in this case showed that there was no true aneurysm present but rather local damage to an atheromatous plaque with partial washout of the plaque. This observation again emphasizes that the angiographic classification of Holmes et al does not adequately consider the underlying pathologic substrate. Similar angiographic images of aneurysm formation after coronary angioplasty have been reported by Hill et al and Weston and Bowerman, and one can only speculate whether or not these would represent the same pathologic correlate.

In each instance in which a dissection was recognized angiographically, extensive laceration of the wall was detected microscopically. Moreover, all such lesions in this series had an eccentric atherosclerotic plaque. It is of interest that the one case diagnosed as a spiral-type dissection was the only one also in which a false channel had occurred within the plaque-free media. The other 5 cases with an extraluminal dissection revealed a dissection that separated the plaque from the underlying medial layers over a considerable distance. Further experiences will have to reveal whether these two angiographic types always correlate with the morphology presented here.

This study is based largely on patients with stable angina pectoris who underwent elective coronary angioplasty. Since the histology of atherosclerotic plaques differs notably between patients with stable angina and those with unstable angina or acute myocardial infarction, one could argue that the angiographic-pathologic correlates at the sites of an emergency angioplasty may be different from those at sites of an elective angioplasty. Further studies are needed to evaluate this possibility. The present observations could serve as point of reference.

References


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