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Anatomic Characteristics of Culprit Sites in Acute Coronary Syndromes

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Abstract

Background—A detailed analysis of the anatomic relationships of the site of culprit lesions that have resulted in acute coronary syndromes (ACS) has not been reported.

Methods—Coronary angiograms of consecutive patients who presented with ACS were analyzed according to multiple anatomic criteria.

Results—In left anterior descending artery (LAD) (n = 85), 85% of culprit lesions were located in the first 40 mm from the ostium. The presence of angulation on the lesion increased the risk of an ACS 1.92 times (95% confidence interval [CI] 1.9–3.07), and the presence of bifurcation after the lesion increased the risk 1.65 times (95% CI 1.04–2.62). Angulated lesions located within the first 40 mm from the ostium and before a bifurcation presented an 11-fold increased risk for an ACS. In right coronary artery (RCA) (n = 58), the risk of plaque rupture was almost 2.5 times higher in lesions located between 10 and 50 mm from the ostium compared to those located in 90–130 mm (relative risk [RR] 2.38, 95% CI 1.25–4.56). In left circumflex (LCx) (n = 40), the risk of plaque rupture was almost 4.5 and 5 times higher in the first 20 mm, and between 20 and 40 mm from the ostium, respectively, compared to 60 and 80 mm (relative risk [RR] 4.58, 95% CI 1.01–20.68 for 0–20 mm, and RR 4.95, 95% CI 1.14–21.47 for 20–40 mm) after adjustment for the presence of curve on the lesion. The presence of lesion angulation increased the risk of plaque rupture almost three times (RR 3.22, 95% CI 1.49–6.93).

Conclusion—Specific anatomic features of the coronary arteries predispose to development and/or subsequent rupture of vulnerable plaques.

Introduction

Coronary plaque disruption with superimposed thrombosis comprises the underlying pathology in acute coronary syndromes (ACS) of unstable angina and myocardial infarction (MI).^{1,2} Several observations have indicated that high-probability zones of coronary thrombosis may exist along the coronary tree.^{3,4} In clinical and pathologic studies, the proximal and mid portions of the major coronary arteries have been identified as the most frequent sites of plaque rupture that have result in ACS.^{5–11}

However, although the geographical distribution of ruptured plaques and coronary thrombosis is well described, data are mainly focused on the distance of the culprit lesion from the coronary ostium. A detailed analysis of the anatomic relationships of the site of culprit lesions that have resulted in ACS has not been reported. Furthermore, most studies have identified the proximal portion of the left anterior descending artery (LAD) as the site of both intravascular ultrasound-detected plaque ruptures and clinical coronary thromboses, but results have been less consistent as far as the left circumflex (LCx) and right coronary artery (RCA) are concerned. Wang et al.¹⁰ reported a predominantly proximal distribution of coronary thromboses in the LCx, whereas Hong et al.,¹² in an intravascular ultrasound study, failed to confirm a predilection for LCx plaque rupture to develop at any specific location. Furthermore, 32% of RCA plaque ruptures occurred at >70 mm from the RCA ostium in that study. This finding was confirmed in patients with both ACS and stable angina pectoris.¹³

We hypothesized that rupture of vulnerable plaques and subsequent development of thrombotic lesions along the coronary tree may depend on several anatomic parameters, such as proximity of side branches and bifurcations, angulation of the relevant coronary segment, and axial bending during the cardiac cycle. All these anatomic features have been correlated with hemodynamic parameters that may potentially affect plaque rupture and subsequent thrombosis.^{3,4} The aim of this study, therefore, was to perform a detailed morphologic analysis of coronary angiograms obtained from patients who presented with unstable angina due to a culprit lesion on a major coronary artery or with anterior ST-elevation MI and a recanalized coronary artery. Our analysis aimed at characterizing lesions that were responsible for ACS by means of distance from the ostium of the artery, proximity of bifurcation(s), diameter of the side branch(es), vessel diameter before and after the lesion, measurement of the angulation of the vessel at the lesion site, and calculation of the degree of axial bending and deformity of the vessel segment that contained the lesion due to ventricular contraction during the cardiac cycle.

Methods

Patients

We analyzed our database with patients who presented in our department with an ACS, i.e., unstable angina or ST-segment elevation MI, and underwent coronary angiography during the period 2002–2005. Unstable angina was defined as precordial chest pain at rest with ST-segment depression on 12-lead electrocardiography (ECG). ST-elevation MI was defined as chest pain associated with ST elevation of ≥ 1 mm in two contiguous ECG leads and subsequent myocardial enzymes rise. Inclusion criteria for the present study were identification of the vessel with the culprit lesion, and a patent vessel, either due to thrombolysis or spontaneous recanalization, at time of angiography. Patients were excluded if they had a previous bypass operation or angioplasty at the site of the culprit lesion. Cases without a clearly identifiable culprit lesion or with multiple significant lesions were also excluded.

Angiographic Analysis

Coronary angiograms were obtained in standard projections after patients had received an intracoronary injection of nitrates. Stenoses were classified according to morphologic criteria as previously described.¹⁴ For quantitative analysis, angiograms were analyzed with the use of a computer-based system (quantitative coronary angiography-coronary measurement system [QCA-CMS] 6.0, Medis, Leiden, the Netherlands). Quantitative coronary angiographic end-points included reference vessel diameter, minimum lumen diameter, and percent diameter stenosis.

In addition, the following anatomic features of lesions were considered:

1. Distance from the ostium of the coronary artery: this was defined as the distance of the part of the lesion with the highest degree of percent diameter stenosis, as defined by QCA, from the ostium of the artery. In order to minimize fore-shortening, standardized angiographic projections, right anterior oblique (RAO) caudal and cranial, were used for measurement of arterial segments.¹⁰
2. Relative position of the side branches: the septal and diagonal side branches were considered for the LAD; the sinus nodal and conus branches, as well as the right ventricular, acute marginal, and posterior descending branches were considered for the RCA; and the left atrial branch, the obtuse marginal branches, and the posterolateral descending branch were considered for the LCx. Vessels were classified according to Bypass Angioplasty Revascularization Investigation (BARI)¹⁵ and Coronary Artery Surgery Study (CASS)¹⁶ coronary artery maps. However, since the anatomic position of these branches displays significant variation, each vessel was considered individually, and the position of each diagonal and septal side branch in relation to the site of the lesion was recorded.
3. Proximity of bifurcation(s), and diameter of the side branch(es): bifurcations were considered relevant if their distance from the culprit lesion was within two prestenotic vessel diameters. The rationale for this choice was the fact that according to computational fluid dynamics (CFD) analysis of coronary flow, shear stress measurements are not affected by bifurcations beyond that distance.¹⁷ For classification of bifurcations, a simplification of the schemes that have been proposed by Lefevre et al.¹⁸ and the SYNTAX¹⁹ (SYNergy between PCI with Taxus and Cardiac Surgery) score was used. Thus, Lefevre type 3 bifurcations (SYNTAX types A and F) were considered as “bifurcations before lesion” for our analysis, whereas Lefevre type 4B (SYNTAX types B and G) were considered as “bifurcations after lesion” (Fig. 1). Lefevre types 1, 2, and 4 bifurcations, i.e., SYNTAX types C and D, were considered as “bifurcations on lesion.”
4. Vessel diameter before and after the lesion: this ratio was used as a measure of shear stress changes.¹⁷
5. Measurement of the angulation of the vessel segment on which the lesion was located, using the application available by the QCA system: this measurement was considered at systole and diastole at the projection that displayed the sharpest curvature of the lesion site (Figs. 2 and 3).
6. Calculation of the degree of axial bending and deformity of the vessel segment that contained the lesion due to ventricular contraction:⁴ this was accomplished by measuring the lesion angle at systole and diastole at the projection that displayed the highest degree of coronary segment flexion. The difference between the two measurements was considered as a measure of lesion deformity due to ventricular contraction during the cardiac cycle (Figs. 4 and 5). For angle measurements, an in-core application in the Medis software was used.

Statistical Analysis

Continuous variables are summarized as mean values $\pm$ standard deviation, and categorical variables as absolute and relative (%) frequencies. Associations among continuous variables were evaluated using the Student's *t*-test, whereas the associations among categorical variables were evaluated through the chi-square test without correction of continuity. To determine whether plaque rupture is equally possible to occur along the relevant coronary artery, we examined whether the observed number of thrombotic lesions were uniformly distributed using one-sample Kolmogorov-Smirnov test. This test compared the observed cumulative distribution function for a variable (i.e., the variable indicating the distance of lesion from the

ostium of the LAD) with a theoretical distribution (i.e., uniform). The binomial test with the probability parameter of 0.5 was used to examine whether plaque rupture frequency is the same with respect to the presence or absence of bifurcation on (before and after) location of lesion, and with the presence or absence of angle on (before and after) location of lesion. This test compares the observed frequencies of the two categories of a dichotomous variable to the frequencies expected under a binomial distribution with a specified probability parameter (0.5). Multiple Poisson regression model was employed to estimate the risk of plaque rupture as a function of distance from the ostium, the presence or absence of curve on the location of culprit lesion, and the presence or absence of bifurcation before and after the lesion. For this purpose, we first divided the lesion's distance from the ostium into 10-mm categories, and we calculated the count of lesions observed in each category, taking into account the presence or absence of curve on lesion and bifurcation before and after lesion. In the Poisson model, the distance from the ostium was entered as a categorical variable with three categories (0–10 mm, 20–40 mm, and 40–60 mm), resulting in the reduction of estimated parameters. Because the presence of bifurcation before lesion was not a statistically significant predictor of the risk of plaque rupture, it was excluded from the final model. A probability value of 5% was considered as statistically significant. All statistical calculations were performed on the SPSS version 13.0 software (SPSS, Inc., Chicago, IL).

Results

Patients

In total, 278 patients with an ACS were identified. Of these, 145 patients were identified with a LAD lesion, 54 patients with a LCx lesion, and 76 patients with a RCA lesion responsible for the clinical presentation. Eighty-five out of 145 patients had an open LAD with at least TIMI (thrombolysis in myocardial infarction) I flow at time of angiography, and were considered for further analysis. Clinical diagnosis was ST-elevation MI in 45 patients, and unstable angina in 40 patients. Forty out of 54 patients in the LCx group, and 58 out of 76 in the RCA group had a patent vessel (LCx and RCA, respectively) with at least TIMI I flow at time of angiography, and were considered for further analysis. Clinical diagnosis was ST-elevation MI in 41 out of 58 patients with a RCA lesion, and in 20 out of 40 patients with a LCx lesion, while unstable angina was diagnosed in the rest of the patients. Table 1 presents clinical characteristics of all patients who were studied.

Analysis of Coronary Angiograms

Characteristics of LAD culprit lesions: In the LAD, in approximately one-third of the patients, the culprit lesion was located before the first septal or first diagonal side branch, and in 70% of patients, lesions were located before the second septal or second diagonal side branch. Culprit lesions were more common within the first 40 mm from the ostium (85% of all cases). Kolmogorov-Smirnov test confirmed the nonuniform distribution of lesions along the LAD ($P = 0.002$).

Table 2 presents the anatomic characteristics of culprit lesions. In the majority of patients (71%), the culprit lesion was located on an angulated vessel segment, as detected in at least one angiographic projection (P for binomial test <0.001). Bifurcations, either before or after the culprit lesion, were also seen in 56% and 66% of cases, respectively (P for binomial test = 0.278 and 0.005 for bifurcation before and after the lesion, respectively). Almost half of the patients (55%) demonstrated axial lesion deformity during the cardiac cycle in at least one projection (P for binomial test = 0.386).

Multiple Poisson regression model showed that the risk of plaque rupture is almost two times and three times higher when the lesion is located within the first 20 mm and between 20 and

40 mm from the ostium, respectively, compared to a location between 40 and 60 mm (relative risk [RR] 2.07, 95% confidence interval [CI] 1.07–4.03 for 0–20 mm, and RR 3.38, 95% CI 1.82–6.28 for 20–40 mm) after adjustment for the presence of curve on the lesion and bifurcation after lesion. Moreover, it was observed that the presence of angulation on the lesion increases the risk almost two times (RR 1.92, 95% CI 1.19–3.07), and the presence of bifurcation after the lesion increases the risk by almost 65% (RR 1.65, 95% CI 1.04–2.62). The risk of plaque rupture for a lesion that displayed a combination of the aforementioned parameters compared to a lesion without these parameters was calculated by multiplying the respective RR reported above. Table 3 illustrates the estimated RR (RR, 95% CI) for all possible combinations of anatomic parameters that were significantly correlated with a ruptured plaque, and were included in the Poisson regression model. The risk of plaque rupture for a lesion located within the first 20–40 mm from the ostium with a curve on the lesion and bifurcation after lesion is approximately 11 times higher compared to that for a lesion located between 40 and 60 mm from the ostium and without any curve or bifurcation after the lesion. In general, the more the anatomic parameters are detected on a lesion, the higher the risk is for plaque rupture and development of an ACS.

Characteristics of RCA culprit lesions: In RCA, 50% of ruptured plaques occurred within the first 50 mm from the ostium. Kolmogorov-Smirnov test confirmed that the lesion's distance from the ostium did not follow the uniform distribution along the RCA ($P = 0.005$).

Table 2 presents the anatomic characteristics of lesions located on RCA. There was a trend for ruptured plaques to occur on angulated vessel segments; in the majority of patients (62%), the culprit lesion was located on an angulated vessel segment, as detected in at least one angiographic projection (P for binomial test = 0.087). Bifurcations, either before or after the culprit lesion, were also seen in 40% and 41% of cases, respectively (P for binomial test = 0.148 and 0.237, respectively).

Forty-seven percent of patients whose culprit lesion was identified on the RCA demonstrated axial lesion deformity during the cardiac cycle in at least one projection (P for binomial test = 0.694). Lesion deformity was more prominent in distal parts of the RCA. We observed that within 10–50 mm, 50–90 mm, and 90–130 mm from the ostium of RCA, 32%, 36%, and 92% of plaque ruptures, respectively, occurred on lesions with axial deformity during cardiac cycle ($P = 0.001$).

In order to estimate the risk of a plaque rupture as a function of distance from the ostium of RCA, the distance of the lesion from the ostium was divided into 10-mm categories, and the number of lesions observed in each category was calculated, taking into account the presence or absence of curve on lesion. Poisson regression was performed by entering in the model the distance of the lesion as a categorical variable with three categories (10–50 mm, 50–90 mm, and 90–130 mm) and the presence of angle on the segment on which the lesion was located. However, the presence of angle was excluded from the final model since it was no more a statistically significant predictor of the risk of plaque rupture. The risk of plaque rupture was almost 2.5 times higher in lesions located between 10 and 50 mm from the ostium compared to those located between 90 and 130 mm (RR 2.38, 95% CI 1.25–4.56), while no difference was observed in the risk of plaque rupture in lesions located between 50 and 90 mm and those located between 90 and 130 mm (RR 1.08, 95% CI 0.50–2.29).

Characteristics of LCx culprit lesions: In the LCx, 82.5% of culprit lesions were located within the first 40 mm from the ostium. Kolmogorov-Smirnov test confirmed that the lesion's distance from the ostium did not follow the uniform distribution along the LCx ($P < 0.001$).

Table 2 presents the anatomic characteristics of lesions located on the LCx. There was a trend for ruptured plaques to occur on angulated vessel segments; in the majority of patients (65%), the culprit lesion was located on an angulated vessel segment, as detected in at least one angiographic projection (P for binomial test = 0.081). Bifurcations, either before or after the culprit lesion, were also seen in 43% and 45% of patients, respectively (P for binomial test = 0.430 and 0.636, respectively). Finally, 68% of patients with LCx lesion demonstrated axial lesion deformity during the cardiac cycle in at least one projection (P for binomial test = 0.038).

In order to estimate the risk of plaque rupture as a function of distance from the ostium of LCx, the distance of the lesion from the ostium was divided into 10-mm categories, and the number of lesions observed in each category was calculated, taking into account the presence or absence of curve on lesion and axial deformity during cardiac cycle. Poisson regression was performed by entering in the model the distance of the lesion as a categorical variable with four categories (0–20 mm, 20–40 mm, 40–60 mm, and 60–80 mm), the presence of angle on the vessel segment on which the lesion was located, and axial lesion deformity during the cardiac cycle. Because the axial lesion deformity was not a statistically significant predictor of the risk of plaque rupture, it was excluded from the final model. The final model showed that the risk of plaque rupture is almost 4.5 and 5 times higher when the lesion is located within the first 20 mm and between 20 and 40 mm from the ostium, respectively, compared to a location between 60 and 80 mm (RR 4.58, 95% CI 1.01–20.68 for 0–20 mm, and RR 4.95, 95% CI 1.14–21.47 for 20–40 mm) after adjustment for the presence of curve on the lesion. Moreover, it was observed that the presence of lesion angulation increased the risk of plaque rupture almost three times (RR 3.22, 95% CI 1.49–6.93).

Discussion

The findings of the present study have identified specific anatomic features that may predispose to development and/or subsequent rupture of an atherosclerotic plaque in all coronary arteries. The majority of culprit lesions on the LAD that result in ACS are located within the first 4 cm from its origin and before the second septal or the second diagonal side branch. In addition, the presence of an angle on the lesion site, as detected in at least one angiographic projection, or a bifurcation before and, particularly, after the lesion are markers of an increased risk for plaque rupture. In RCA, half of the ruptured plaques were within the first 50 mm from the ostium, whereas in LCx, the majority of ruptured plaques (82.5%) occurred within the first 40 mm from the ostium. Lesion angulation denotes an increased risk of plaque rupture in the LCx, and axial lesion deformity due to left ventricular contraction is seen in 92% of ruptured plaques in the distal part of RCA. Although the geographical distribution of thrombosis on the coronary arteries has been previously described, data are mainly focused on the distance of the culprit lesion from the coronary ostium. A detailed analysis of the anatomic relationships of the site of culprit lesions that resulted in ACS has not been reported.

The spatial distribution of acute MI occlusions has been previously reported. In 1987, el Fawal and colleagues,⁵ in a pathologic study on 59 patients who died of MI in Glasgow, provided evidence that thromboses are distributed in the proximal coronary vessels. Similar observations were subsequently published by other groups.^{6–9} In young subjects less than 40 years of age, dying of noncardiovascular causes, coronary plaques are concentrated proximally, and diminish with distance in all coronary arteries.⁶ These observations suggest that proximal coronary plaques are more prone to rupture. Recently, Wang and colleagues¹⁰ demonstrated that acute coronary occlusions leading to MI tend to cluster in predictable “hot spots” within the proximal third of the coronary arteries, particularly the LAD and LCx. Recent pathologic studies have also demonstrated that over 50% of thin-cap fibroatherosclerotic plaques occur in the proximal portions of the major coronary arteries, another one-third in the mid portion of these arteries, and the rest are distributed in distal segments.¹¹ Valgimigli et al., in intravascular

ultrasound studies, have shown that plaque distance from the coronary ostium is an independent determinant of relative lipid content.^{20,21} Thus, plaques located within the proximal 20 mm of coronary vessels are relatively richer in lipid content compared to those more distally located,²⁰ and this is also true for main stem lesions.²¹ Our results are in keeping with these observations, and support the notion of a specific distribution pattern of ruptured plaques that results in ACS.

A higher incidence of ruptured plaques was detected before and after bifurcation in the LAD, and at sites of vessel angulation in the LAD and LCx. These findings are interesting, particularly in view of the emerging evidence that relates parameters, such as shear stress and mechanical stretching, with regional vessel morphology. Several studies have now validated the low shear stress hypothesis of atherosclerosis,^{22–24} and have revealed that atherogenesis preferentially involves the outer walls of vessel bifurcations, side branches, and the inner walls of regions of high curvature in the arterial tree.^{23–26} In these geometrically predisposed locations, vessel wall shear stress is significantly lower in magnitude, and exhibits directional changes and flow separation, features absent from regions of the arterial tree that are generally spared from atherosclerosis.^{25–30} Indeed, in the straight parts of the vascular system, the blood flow velocity profile exhibits a parabolic shape, with the velocity being maximum at the center of the vessel and zero at the vessel walls. Since wall shear stress is defined as the product of the dynamic blood viscosity and the rate of blood flow velocity change near the vessel wall (namely, the wall shear rate),¹⁷ in the straight parts of the vascular system, wall shear stress is equal in all segments of the vessel wall. In curved segments of the vascular system, the blood flow velocity profile is skewed toward the outer wall of the curved vessel, i.e., the blood velocity in the outer vessel wall is greater than the velocity in the inner wall with respect to the curvature.^{30,31} In this case, wall shear stress is no longer equal in all segments of the vessel wall, but is maximum in the outer vessel wall and minimum in the inner vessel wall with respect to the curvature. Although the relationship between wall shear stress and development of atherosclerosis seems rather established, its potential effect on plaque rupture has not been studied extensively. The magnitude of wall shear stress is inversely proportional to the cube of the artery radius.³⁰ Therefore, a small change in the radius of the artery, as usually happens following the emergence of a side branch, will produce a large change in wall shear stress. In reality, flow into a stenotic zone will increase wall shear stress more than that anticipated by the change of radius alone because the acceleration of blood into a stenosis flattens the parabolic velocity profile, leading to a much more rapid increase in blood velocity adjacent to the vessel wall, and thus increased wall shear stress.³² It seems that the wall shear stress at a 50% subcritical stenosis is within the range of shear forces hypothesized to be capable of inducing endothelial damage.³³ The fact that shear forces induce marked endothelial damage does not prove that they are capable of causing plaque rupture. However, intimal damage, even if initially of minimal mural depth, may increase the likelihood of rupture at sites of increased wall shear stress.³⁴ Observations of regression of graft hyperplasia stimulated by high shear stress in nonatherosclerotic baboons indicate that high shear stress stimulates the endothelium to induce thinning of the fibrous cap.³⁵ Thus, areas with anatomy that favors acute changes in shear stress may well be predisposed to higher possibility of atherosclerotic plaque rupture.

The effect of local mechanical stress on the normal and diseased moving arterial wall with lipid pool and plaque cap has been investigated in a patient-specific three-dimensional coronary arterial tree reconstruction by finite-element analysis.³⁶ A large value of stress gradient was found at bifurcation regions, indicating that higher stress occurs at junction between the proceeding and branching vessels during cyclic flexion.³⁶ Additionally, the regions closer to the bifurcations underwent a larger deformation, yielding a higher stress, than those further to the bifurcations. It seems that when the length of the lesion increases by 100%, the maximum plaque stress increases by 10–30%, and the stress concentration becomes less localized,

whereas when the diameter of the vessel is reduced to half of its size, the maximum stress dramatically increases by 300–400%, and the mechanical stress gradient becomes higher.³⁷

In the distal part of the RCA, 92% of ruptured plaques were located on lesions that displayed detectable deformation and flexion during the cardiac cycle. Coronary arteries tethered to the surface of the beating heart undergo cyclic longitudinal deformations by axial bending (flexion) and stretching.^{4,38} Approximately 40 million flexions of the coronary arteries occur during 1 year.³⁸ In the RCA, coronary motion does not appear to significantly affect shear stress measurements,³⁹ but may extend mechanical distortion of atherosclerotic plaques. Like circumferential bending, a sudden accentuated longitudinal flexion may trigger plaque disruption, whereas long-term cyclic flexion may fatigue and weaken the plaque.⁴ Perhaps additional angiographic projections might have revealed further arterial segments that are subjected to axial deformity throughout the cardiac cycle.

Study Limitations

The main limitation of our study is that our analysis has been conducted on two-dimensional coronary angiograms that may not allow full assessment of spatial anatomic relationships. Second, occluded vessels were not considered, since it was impossible to obtain an accurate depiction of the vessel on the culprit site and beyond it, and primary angioplasty might have introduced morphologic changes, particularly when a stent was used. This, however, might have unavoidably introduced selection bias.

In conclusion, our results support the emerging evidence that the risk of plaque disruption is related to intrinsic properties of individual plaques (segment vulnerability), as well as extrinsic forces acting on plaques (rupture triggers). The former predispose plaques to rupture, whereas the latter may precipitate disruption if vulnerable plaques are present. We have previously discussed the concept of vulnerable coronary segment in the context of ACS.⁴ Potential identification of these high-risk zones along the coronary tree should be of paramount clinical importance for locally directed preventive strategies.^{40,41} Our data provide the rationale for the development of risk stratification tools for the characterization of coronary stenoses and plaques that are diagnosed on the LAD at coronary angiography. Further in vitro and in vivo studies of human coronary anatomy, as well as in-depth analysis of coronary hemodynamics, are necessary for characterization and identification of such vulnerable segments.

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RAO Cranial

Figure 1.
Typical angiographic appearance of a ruptured plaque from a patient with unstable angina and mild enzyme elevation. A “postlesion” bifurcation is seen immediately after the plaque (arrow).

RAO Cranial **LAO Caudal****Figure 2.**

Typical angiographic appearance of a ruptured plaque from a patient with an acute anteroseptal MI. In RAO cranial projection (left panel), no significant angulation is seen at the lesion site. In LAO caudal projection (right panel), a sharp angle on the lesion is revealed. This projection was considered for measurement of the lesion angle. In this case, bifurcations are seen “pre” and “post” the lesion.

Figure 3.
Typical angiographic appearance of a ruptured plaque from a patient with an acute lateral MI. In RAO caudal projection (left panel), no significant angulation is seen at the lesion site on the LCx. In RAO cranial projection (right panel), a sharp angle on the lesion is revealed. This projection was considered for measurement of the lesion angle.

Figure 4. Deformity of a LAD lesion induced by ventricular contraction during the cardiac cycle, as seen in the LAO cranial projection. At diastole (left panel), the angulation is 160.5°, whereas in systole (right panel), this angle becomes 136.1°.

Figure 5. Deformity of a RCA lesion induced by ventricular contraction during the cardiac cycle, as seen in the LAO projection. At diastole (left panel), the angulation is 175.1° , whereas in systole (right panel), this angle becomes 159.7° .

Table 1

Clinical Characteristics of Studied Patients with LAD, RCA, and LCx Lesions

	Patients with LAD Lesions (n = 85)	Patients with RCA Lesions (n = 58)	Patients with LCx Lesions (n = 40)
Male gender	72 (85%)	54 (93%)	37 (93%)
Age (years)	57.8 ± 11.5	53.00 ± 1.37	55.60 ± 1.72
Diabetes mellitus	19 (23%)	12 (21%)	3 (7.5%)
Hypertension	40 (50%)	26 (45%)	18 (45%)
Hypercholesterolemia	57 (70%)	37 (64%)	31 (18%)
Smoking	54 (66%)	46 (79%)	29 (73%)
Family history of IHD	7 (9%)	9 (16%)	5 (13%)
Enzyme rise	60 (71%)	45 (78%)	30 (75%)
Diseased vessels			
1	47 (55%)	27 (46%)	14 (35%)
2	28 (33%)	23 (40%)	15 (38%)
3	10 (12%)	8 (14%)	11 (28%)

IHD = ischemic heart disease.

Table 2
Anatomic Characteristics of Culprit Sites on LAD, RCA, and LCx

Anatomic Parameters	LAD n = 85	RCA n = 58	LCx n = 40
Distance from the ostium (mm)	26.5 ± 12.1	58.57 ± 30.52	29.73 ± 14.39
Stenosis (%)	68.5 ± 11.2	65.86 ± 14.98	61.15 ± 14.36
Vessel diameter before lesion (mm)	3.1 ± 0.7	3.01 ± 0.74	2.86 ± 0.49
Vessel diameter after lesion (mm)	2.9 ± 0.7	2.90 ± 0.81	2.71 ± 0.50
Bifurcation (y/n)			
<i>Before lesion</i>	48 (56%)	23 (40%)	17 (43%)
<i>After lesion</i>	56 (66%)	24 (41%)	18 (45%)
Diameter of bifurcation (mm)			
<i>Before lesion</i>	1.6 ± 0.6	1.18 ± 0.43	1.38 ± 0.83
<i>After lesion</i>	1.5 ± 0.6	1.18 ± 0.49	1.26 ± 0.48
Difference on lesion angle between systole and diastole (y/n)	47 (55%)	27 (47%)	27 (68%)
Axial deformity (degrees)	15.8 ± 7.6	27.71 ± 10.42	25.74 ± 13.35
Angulation on lesion (y/n)	60 (71%)	36 (62%)	26 (65%)
Degrees of angulation on lesion	127.4 ± 26.5	116.94 ± 30.57	133.54 ± 26.72

Table 3

Estimated Relative Risk of Plaque Rupture in the LAD (RR, 95% CI) for All Possible Combinations of Anatomic Parameters Included in the Poisson Regression Model

Distance from the Ostium (0–20 mm)	Distance from the Ostium (20–40 mm)	Curve on Lesion	Bifurcation after Lesion	RR (95% CI)
Yes	–	Yes	Yes	6.6 (2.5–18.2)
Yes	–	Yes	–	4.0 (1.8–8.9)
Yes	–	–	Yes	3.5 (1.5–7.7)
–	Yes	Yes	Yes	10.7 (4.22–27.4)
–	Yes	Yes	–	6.5 (2.9–14.2)
–	Yes	–	Yes	5.6 (2.6–12.2)

RR illustrates the risk of plaque rupture for a lesion that displayed a combination of the anatomic parameters compared to a lesion without these parameters (reference group for distance of lesion from the ostium is 40–60 mm).