Comparison of Intravascular Ultrasound and Angiography in the Assessment of Myocardial Bridging

Junbo Ge, MD; Raimund Erbel, MD; Hans-Jürgen Rupprecht, MD; Lothar Koch, PhD; Peter Kearney, MRCP; Günter Görge, MD; Michael Haude, MD; Jürgen Meyer, MD

Background In autopsy, myocardial bridging is a common finding. With coronary angiography, a systolic compression, mainly of the left anterior descending coronary artery, is observed in 1% to 3% of the patients. Controversy exists concerning the functional importance of this finding. To obtain a functional insight into the myocardial bridging, intravascular ultrasound and intracoronary Doppler were performed.

Methods and Results Intracoronary ultrasound and Doppler were performed in 14 patients with angiographic evidence of systolic vessel compression ("milking effect") in the left anterior descending coronary artery. The 4.8F, 20-MHz ultrasound catheter could not be advanced through the entire myocardial bridge segment in 6 of the 14 patients studied because the lumen was <1.6 mm. In these patients, only the proximal parts of the bridge segment were scanned. The changes in cross-sectional shape during the cardiac cycle were determined for both the normal proximal segment and the bridge segment by use of a semiautomatic computer program. Intracoronary Doppler (20 MHz) was performed in 7 patients with a 3F catheter. A highly characteristic systolic eccentric or concentric compression with delayed relaxation in diastole of the myocardial bridging segment was clearly visualized in all patients. The cross-sectional lumen area variation was 40±25% in the bridging segments and 9±7% in the normal segments (P<.01). No atherosclerotic lesions were detected in the bridge or the distal segment in the 8 patients in whom the IVUS catheter was successfully advanced through the entire myocardial bridge. However, atherosclerotic plaques were found in the segments proximal to the bridge in 12 of 14 patients (86%). The resting mean flow velocity was 6.4±1.2 cm/s; the maximal mean flow velocity after intracoronary administration of 10 mg papaverine was 14.1±3.4 cm/s. The coronary flow reserve was 2.2±0.7. A highly characteristic pattern showing a prominent peak in coronary velocity in early diastole was observed in 86% of patients, and this pattern was enhanced after injection of intracoronary papaverine.

Conclusions Intravascular ultrasound demonstrated a characteristic systolic compression of the bridge segments. The delayed compression release may explain the characteristic sharp early diastolic peak in coronary flow velocity found with intracoronary Doppler in vessels with myocardial bridging. Reduced coronary flow reserve may be related to this phenomenon, possibly explaining signs of ischemia detected in some of the patients, but may alternatively be a result of the presence of atherosclerosis in the segment proximal to the bridge in these patients. (Circulation. 1994;89:1725-1732.)

Key Words • ultrasonics • myocardium • atherosclerosis • coronary flow

Coronary arteries and their major branches usually course on the surface of the heart in the subepicardial tissue. Myocardial bridging is said to occur when a segment of an artery travels through the myocardium. This phenomenon was first described by Grainiciuan in the early 1920s.1 In 1960, Portmann and Twigg2 first reported the radiological appearance of transient occlusion in a segment of the left anterior descending coronary artery during systole. A large discrepancy exists between pathological series, in which the incidence has varied from 15% to 85%,3,4 and angiographic series, in which it is reported as being between 0.51% and 2.5%.5-7 Reports have suggested that myocardial bridging may be associated with myocardial ischemia,8-10 myocardial infarction,11-13 conduction disturbances,14 and sudden death.15,16 Although the systolic compression can be enhanced by nitroglycerin and positive inotropic drugs,17,18 no signs of ischemia during stress testing have been detected by thallium scintigraphy.19 Nevertheless, Noble and colleagues3 observed that patients with a severe milking effect (more than 75% narrowing during systole) can experience marked myocardial ischemia at rapid heart rates, as evidenced by ST segment depression and increased left ventricular lactate products. Intravascular ultrasound imaging (IVUS) is a new technique that allows accurate assessment of vascular anatomy. Preliminary reports have shown that this technique is able to detect myocardial bridging and provide unique information concerning wall morphology in this condition.20,21 The purposes of this study were (1) to evaluate the morphology of myocardial bridging by intravascular ultrasound, (2) to assess the time course of compression and the compression release of the myocardial bridge segment in comparison with the normal segment, and (3) to determine the coronary flow velocity reserve.

Methods

Patients

Fourteen patients (12 men, 2 women) with typical or atypical angina who had undergone diagnostic coronary angi-
ography and in whom an angiographically demonstrated milking effect of the left anterior descending coronary artery had been seen were selected for IVUS examination. The patients ranged in age from 47 to 69 years (mean ± SD, 58 ± 9 years). All gave informed consent. None of them had previous myocardial infarction or left ventricular hypertrophy evaluated by echocardiography.

IVUS Device

The IVUS catheter used in this study consists of a 4.8F or 3.5F catheter and a 20-MHz ultrasound transducer inside the catheter (Sonicaid, Boston Scientific Corp, Watertown, Mass). The transducer is mechanically rotated within the catheter at 600 to 800 rpm to provide cross-sectional images via an ultrasound diagnostic imaging console (Diasonic, Milpitas, Calif). An ECG is recorded simultaneously. The images are recorded on 1/2-in super VHS videotape for off-line analysis.

Intracoronary Doppler Device

The intracoronary Doppler catheter used is a commercially available 3F catheter with an end-mounted transducer (model DC-201, Millar Instruments Inc). The signal is generated by a pulsed transmitter-receiver (Mikro-Tip, Millar). Operating frequency is 20 MHz and the pulse repetition rate is 62.5 kHz. The Doppler shift is analyzed by zero-crossing techniques, resulting in mean and phasic signals.

Techniques

Each patient received 20 mg of isosorbide dinitrate and 10 mg nifedipine orally before catheterization. Before each IVUS examination, the patient received 3000 IU of intracoronary heparin. An 8F or 9F giant lumen guiding catheter (Medtronic) was used to position the ultrasound catheter for IVUS imaging. A 3-m, 0.014-in guide wire was placed in the desired vessel, and the IVUS catheter was advanced by use of a monorail technique to obtain coaxial images. The IVUS catheter was then drawn back slowly while the images were recorded in real time and stop frames. The position of the IVUS probe was documented and numbered on x-ray film at the points where the IVUS images were taken. After IVUS examination, the catheter was removed but the guide wire left in situ in the coronary artery. The Doppler catheter was then advanced over the wire to the proximal portion of the bridge segment. The position chosen was dependent on obtaining a high-quality phasic signal of blood flow velocity. After the resting velocity was recorded, 10 mg papaverine was administered into the coronary artery via the guiding catheter during continuous recording. An estimation of the coronary flow reserve was obtained by calculating the ratio between the maximal mean flow velocity after the administration of intracoronary papaverine and resting mean flow velocity.

Data Analysis

IVUS Image Analysis

All the IVUS images were digitized into a 512×512-pixel matrix with an image processing computer (Echo-Com, PPG Hellige, Germany) that enables digitization of a series of 32 frames of IVUS images. The images were stored on a 1-giga-byte erasable optical disk (Mxtor, the Netherlands). These images were reviewed; a segment of artery was considered normal when no systolic compression of the lumen was evident and in accordance with the criteria proposed by Nissen et al. The frames showing the maximal and minimal vessel areas of the myocardial bridge segment were magnified by use of the built-in zoom function, and the cross-sectional area was determined by planimetry. The values were then compared with the measurements derived from angiography. The cross-sectional plaque area was quantified by outlining the area of the lumen and the area of the plaque at the boundary of the media/plaque interface. Subtraction of the lumen area from the total area yielded the cross-sectional area of the plaque. Circumferential compression of the vessel was considered concentric; compression was termed eccentric when parts of the vessel circumference were not affected.

Ninety consecutive frames of videotaped IVUS images of the myocardial bridge segment and of the normal segment proximal to myocardial bridging were digitized into a 256×256-pixel computer. A semiautomatic software based on PWAVE (Precision Visuals, Inc, Boulder, Colo) was developed for the evaluation of the lumen area. PWAVE is a computer program for analyzing and displaying scientific data. The evaluation was performed on a SUN SPARC-2 workstation (Mountain View, Calif). The program compensates for catheter movement caused by heartbeat by centering each image on consecutive frames. A region of interest comprising both the lumen and the vessel wall is defined interactively. Both the vessel wall and lumen were segmented by thresholding. To control the segmentation process, the determined contours of the lumen were superimposed on the original images. In case of mismatch, resulting, for example, from signal dropout, the procedure was repeated after the threshold or the region of interest was corrected for. The area of the segmented lumen was measured after removal of the catheter and guide-wire artifact. The pulsatile variation of the cross-sectional lumen area of at least three cardiac cycles was displayed. For each cardiac cycle, the maximum area, the minimum area, and the end-diastolic area as identified by ECG were determined.

Angiographic Measurement

The cineangiograms of the myocardial bridge segment and the normal segment proximal to the myocardial bridging in single right anterior oblique 30° view were projected and magnified with a CAP-35B angiogram projection system (Weinberger AG, Zürich, Switzerland). Both the normal segment and the systolic and diastolic frames of the myocardial bridge segment were fed into a 256×256-pixel matrix (Kontron Cardio 500, Eching, Germany). The lumen diameters were measured manually with the guiding catheter as a reference. The vessel area was calculated from the derived diameter. Fig. 1 illustrates the angiogram and quantitative analysis of a myocardial bridge segment in systole (A) and diastole (B).

Statistics

All the data were analyzed by the PRIMER STATISTICS program (McGraw-Hill). Results of the measurements of the lumen area, lumen area variation, and coronary flow reserve are expressed as mean ± SD. The cross-sectional dimensions of the normal segment and the myocardial bridge segment derived from IVUS and angiography at corresponding sites were analyzed by linear regression. The maximal and minimal dimensions of the myocardial bridge segment during a cardiac cycle were compared from IVUS and angiography were compared by paired t tests. A probability value of P<.05 was considered statistically significant. The reproducibility of IVUS imaging for measuring coronary vessel dimensions in the myocardial bridge segment is presented as intraobserver and interobserver correlation coefficients. Intraobserver (J.G.) and interobserver (J.G. and P.K) correlation coefficients for measuring the lumen diameter were 0.996 and 0.99; the standard errors of the estimates (SEE) were 0.08 and 0.11 mm. Intraobserver and interobserver correlation coefficients for measuring lumen area were 0.996 and 0.988; SEEs were 0.32 mm² and 0.51 mm².

Results

Morphological Observation

The IVUS catheter could not be advanced through the entire myocardial bridge segment in 6 of the 14 patients (43%) studied because the lumen diameter was
<1.6 mm. In these patients, only the proximal parts of the bridge segment were scanned. A highly characteristic systolic compression with delayed relaxation in diastole of the bridge segment was clearly visualized in all patients. Fig 2 shows the lumen shape change in a bridge segment during a cardiac cycle. The lumen area decreased from 11.3 to 6.1 mm²; the lumen diameter decreased from 3.8 to 2.8 mm. The lumen compression
was observed as being eccentric in 12 of 14 patients (86%) and concentric in 2 of 14 patients (14%). In 12 patients, atherosclerotic plaque was found in the segments proximal to the bridge (Fig 3), and in 4 of these patients, percutaneous transluminal coronary angioplasty (PTCA) was subsequently undertaken. The maximal plaque area ranged from 2 to 12 mm² (mean±SD, 6.9±3.9 mm²). No atherosclerotic lesions were detected in the myocardial bridge segments or the segments distally in the 8 patients in whom the IVUS catheter was successfully advanced through the entire bridge segment.

Quantitative Analysis of the Myocardial Bridge and the Normal Segment Proximal to the Bridge

The most compressed segment that could be reached was selected to analyze the IVUS measurements. Table 1 lists the correlations for the lumen dimensions be-

<table>
<thead>
<tr>
<th>Coronary Segment</th>
<th>Regression</th>
<th>r</th>
<th>P</th>
<th>SEE</th>
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<tr>
<td>D</td>
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<td>.01</td>
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<tr>
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<td>y = 1.03x + 2.14</td>
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<td>.01</td>
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<tr>
<td>Myocardial bridging</td>
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<tr>
<td>Diastole</td>
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<tr>
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<td>A</td>
<td>y = 1.02x + 2.02</td>
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<td>.02</td>
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<td>Systole</td>
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<tr>
<td>D</td>
<td>y = 3.63 - 0.69x</td>
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<td>A</td>
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<td>-.37</td>
<td>.36</td>
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D indicates lumen diameter; A, lumen area.
Table 2. IVUS, Angiography, and ICD Measurements

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<td>A, mm²</td>
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<td>2.58</td>
<td>5.00</td>
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<td>2.95</td>
<td>0.46</td>
<td>1.80</td>
<td>3.9</td>
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</table>

IVUS indicates intravascular ultrasound; ICD, intracoronary Doppler; D, lumen diameter; A, lumen area; RFV, resting flow velocity; MFV, maximal flow velocity; and CFR, coronary flow reserve.

Changes are illustrated in Fig 4, where the largest lumen of the selected myocardial bridge segment is seen in mid diastole and the smallest lumen between end systole and early diastole (Fig 4B). The cross-sectional lumen area variation during the cardiac cycle was 40±25% in the myocardial bridge segments and 9±7% in the normal segments (P<.01) (Fig 4).

Coronary Flow Reserve

Coronary flow reserve was available in 7 of 14 patients. The resting flow velocity was 6.4±1.2 cm/s; the maximal flow velocity was 14.1±3.4 cm/s. The calculated coronary flow reserve was 2.2±0.7 (Table 2). A highly characteristic pattern showing a prominent peak in coronary flow in early diastole was observed in 6 of 7 patients (86%) (Fig 5). This characteristic flow pattern was abolished as the Doppler transducer was pulled back to the normal segment (Fig 6).

Discussion

Since Portmann and Iwig2 described angiographically visualized systolic narrowing of the left anterior descending coronary artery, many subsequent reports have described the milking effect of this artery. The milking effect was thought to be caused by systolic compression of the intramural segment, which was seen in conjunction with various cardiac conditions, including coronary artery disease, left ventricular hypertrophy, and hypertrophic cardiomyopathy.3,5,6

In the present study, in which all the patients had typical or atypical angina, angiography demonstrated stenoses in only 4 patients, in whom PTCA was subsequently undertaken. In contrast, IVUS identified 12 of 14 patients with atherosclerotic plaques in the segments.
proximal to the myocardial bridging. In the myocardial bridge segment and in the segment distal to it, no plaque was detected. This finding is consistent with the pathological observations that atherosclerotic lesions seldom involve the bridge segment itself.27,28 The reason for this phenomenon is unknown, but it may relate to a pattern of wall stress and subsequent injury to which the segment proximal to the myocardial bridging is particularly prone.29

Significant differences were obtained between the cross-sectional area variation derived from IVUS and angiography during the cardiac cycle. The analysis of IVUS-derived measurements systematically excludes cases in which the catheter could not be passed as the result of a lumen diameter of <1.6 mm for much of the cardiac cycle. Maximal systolic compression in the myocardial bridging in some cases may have been limited by the IVUS catheter. These factors inevitably increase the mean minimum lumen measurements obtained on IVUS and may in large part explain the discrepancy with the angiographic findings. Smaller IVUS catheters would address this problem. The accuracy of quantification of a single plane area on the basis of the angiograms in a condition resulting in an invariably nonspherical lumen is certainly limited and may also explain the discrepancy we found. The better correlations of the dimensions in diastole than systole support this conclusion.

Risse and Weiler30 reported that the intima of the myocardial bridge segment (66.3 μm) was thinner than that of the proximal part (406.6 μm). In this study, we were not able to determine the thickness of the intima and media of the myocardial bridge segment because the resolution of the 20-MHz IVUS catheter we used is too low at approximately 150 μm.

For the first time in a large group of patients, we measured coronary flow velocity to calculate coronary
flow reserve. A reduction in coronary flow reserve was demonstrated in patients with myocardial bridging. This may be due to a moderate increase in resting flow velocity and a limited hyperemic response as a result of reduced mid to late diastolic flow velocity and marked reduction in systolic flow velocity, as demonstrated in Fig 5. These results may help to explain the previous observations that this syndrome is associated with myocardial ischemia, myocardial infarction, and conduction system disturbances.5,6,16 Alternatively, the reduced coronary flow reserve may be a reflection of the presence of atherosclerosis in the segment proximal to the myocardial bridging.

A typical Doppler pattern showing an early prominent diastolic peak in coronary flow velocity is found in myocardial bridge segments, as illustrated in Figs 5 and 7. A sharp acceleration of flow is seen in early diastole, followed by immediate marked deceleration. Flow velocity plateaus for the remainder of diastole before a drop in systole. In contrast to the normal pattern of vessel area change during the cardiac cycle, in which an increase in area is seen throughout systole in conjunction with the rise in aortic root pressure, myocardial bridge segments undergo a marked decrease in area, particularly in the second half of systole. Importantly, this vessel compression persists into early diastole. The hemodynamic consequences of these changes in vessel area can be seen in the coronary flow velocity traces in the schematic drawing in Fig 7. In early diastole, as the intraventricular pressure quickly drops during the slower decline in aortic root pressure, the maximal coronary perfusion pressure occurs. In normal coronary arteries, this results in a typical acceleration in coronary flow followed by a gradual decline. The rate of deceleration may be attenuated by the simultaneous gradual decrease in vessel area. In contrast, the very sharp increase in velocity in the bridge segment is the result of maximum perfusion pressure coinciding with a persistently contracted vessel area, giving rise to a steep pressure gradient across the bridge. The sudden deceleration appears to be the result of the rapid increase in vessel area that occurs during relaxation of the bridge in diastole, which abolishes the pressure gradient. This typical pattern was found in 86% of our patients with myocardial bridging who underwent Doppler examination. The atypical flow pattern may be a result of the position of the Doppler catheter, which was placed too far proximal to the myocardial bridging, as previously reported.21 Although the structural and functional data acquired in this study strongly support such an explanation, simultaneous intracoronary pressure measurements are needed to corroborate the hypothesis.

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