Frequency of Myocardial Bridges and Dynamic Compression of Epicardial Coronary Arteries
A Comparison Between Computed Tomography and Invasive Coronary Angiography

Pyung Jin Kim, MD; Gham Hur, MD, PhD; Su Young Kim, MD; June Namgung, MD; Seong Wan Hong, RT; Yong Hoon Kim, MD; Won Ro Lee, MD

Background—The objective of the present study was to describe the relative frequency of myocardial bridging and dynamic compression of the coronary arteries as assessed by CT angiography and conventional angiography.

Methods and Results—A total of 311 patients (208 men, 103 women; mean age 63 years) who received both 64-section CT angiography and conventional angiography were reviewed retrospectively for myocardial bridging of the left anterior descending coronary artery. Myocardial bridging was considered when the left anterior descending coronary artery was within the interventricular gorge and was classified as either myocardial bridging with partial encasement or myocardial bridging with full encasement, according to the extent of vessel encasement by the myocardium. The length, location, and depth of myocardial bridging were correlated with the presence, length, and degree of dynamic compression observed at conventional angiography. Among the 300 patients studied (11 were excluded), myocardial bridging was observed at CT angiography in 174 (58%) as partial encasement (n=77) or full encasement (n=117). Conventional angiography revealed dynamic compression in 40 patients (13.3%; partial encasement in 1 patient and full encasement in 39). The length of the dynamic compression was considerably longer than the respective tunneled segment in all patients. Total length correlated (P=0.003) with the dynamic compression, but depth did not (P=0.283).

Conclusions—The frequency of myocardial bridging observed by CT angiography was 58%, and conventional angiography revealed dynamic compression in 13.3% of total patients. Dynamic compression occurred almost exclusively (97.5% of the time) in patients with full encasement of the left anterior descending coronary artery, regardless of the presence of overlying muscle. (Circulation. 2009;119:1408-1416.)

Key Words: coronary disease myocardium heart defects, congenital angiography tomography

Myocardial bridging, an inborn abnormality, is defined as the presence of an intramyocardial segment of a major coronary artery that normally has an epicardial course (Figures 1 and 2).1 Myocardial bridging is clinically significant when associated with regional hemodynamic alterations, and studies have shown that such instances of myocardial bridging are linked to clinical complications that include ischemia, acute coronary syndrome, coronary spasm, arrhythmia, and sudden death, although in the vast majority of cases, myocardial bridging remains clinically silent.1-5 Because the clinical significance of myocardial bridging lies in the related regional hemodynamic changes that occur almost exclusively in the left anterior descending coronary artery (LAD),2,4,6 the depiction of myocardial bridging in the LAD that is likely to cause dynamic compression appears to be clinically important.

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From the Departments of Radiology (P.J.K., G.H., S.Y.K., S.W.H., Y.H.K.) and Cardiology (J.N., W.R.L.), Inje University Ilsanpaik Hospital, Goyang-si, Korea.
Correspondence to G. Hur, MD, PhD, Inje University Ilsanpaik Hospital, 2240 Daewha-dong, Ilsanseo-gu, Goyang-si, Gyeonggi-do, Korea. E-mail ghuster@gmail.com
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The wide discrepancy in the reported prevalence of myocardial bridging between autopsy findings (average 33%, range 15% to 85%1,2,7-11 and those of conventional angiography (CA; average 5%, range 0.5% to 16%)2,3,11-16 has been attributed to myocardial bridging that does not cause dynamic compression (milking effect), a characteristic angiocraphic finding of myocardial bridging.3 Recently, CT angiography (CTA) has been used in the diagnosis of myocardial bridging, with an average depiction rate between that of autopsy and CA but with a wide variation among authors (average 21%, range 3.5% to 38.5%).17-20

The wide variation in the prevalence of myocardial bridging in CTA among authors, together with our new
observation of dynamic compression in patients with no overlying muscle (Figures 3 and 4), prompted us to seek improved diagnostic criteria for the detection of myocardial bridging by CTA. The objective of the present study was to assess the frequency of myocardial bridges and dynamic compression of the coronary arteries using advanced coronary CT imaging and conventional coronary angiography.

Figure 1. CTA showing normal epicardial course of the LAD in a 63-year-old woman. The 5-mm-thick long-axis curved multiplanar reformation image (left) of the LAD reveals an epicardial location. Visualization of the septal branches (arrowhead) entering the septum indicates the optimal angle of the plane. Short-axis images (right), obtained at 0.5-mm thickness with a gap of 0.9 mm perpendicular to the long axis of the LAD, depict this vessel at some distance from the interventricular gorge (g). A septal branch arising from the proximal segment of the LAD (arrows) travels deep into the septum along its outer margin. The LAD is at the center of the short-axis images. RV indicates right ventricle; LV, left ventricle.

Figure 2. Classification of vessel encasement by myocardium. A, Schematic. B, Short-axis CTA images obtained perpendicular to the long axis of the LAD (center of the image) demonstrate the location of the LAD (white dot) in normal epicardial space at some distance from the interventricular gorge (far left). Myocardial bridging with partial encasement is defined as the LAD being within the interventricular gorge and in direct contact with left ventricular myocardium. Myocardial bridging with full encasement is defined as the LAD being surrounded by the myocardium but without measurable (>0.7 mm; third from left) or with measurable (far right) overlying muscle. Display window/level settings of 110/85 HU (top of B) and 256/128 HU (bottom of B) were used. S indicates septum; RV, right ventricle; and LV, left ventricle.
Methods

Patients
Of 2280 consecutive patients who underwent CTA between June 2006 and July 2007, 311 (208 men, 103 women; mean age 63 years) who also underwent CA for clinical indications were reviewed retrospectively for the presence of myocardial bridging. Eleven were excluded because of total occlusion (n=3), bypass surgery (n=3), or artifacts (n=5; Figure 5).

CT Coronary Angiography
The preparation of patients included an oral dose of 50 to 75 mg of atenolol if the heart rate was >65 bpm 1 hour before examination and sublingual nitroglycerin (0.3 mg) immediately before scanning. Patients with uncontrollable arrhythmia or tachycardia (heart rate >80 bpm) and those who cooperated poorly were excluded. After prospective precontrast scanning, an Aquillion 64 (Toshiba Medical Systems, Tokyo, Japan) was used to retrospectively obtain ECG-gated spiral postcontrast scans (0.5-mm section thickness; pitch of 0.225 to 0.24; 0.4-second rotation time; 180-cm field of view; and 120-kV tube potential). Tube current was modulated individually with the SD of CT numbers obtained during precontrast scanning at the level of the left atrium.21
The automatic triggering system was activated by the presence of 140 to 160 Hounsfield units (HU) at the aortic root after the injection of 60 to 80 mL (scaled according to body weight) of nonionic contrast at a rate of 4.5 mL/s, chased by 30 mL of normal saline. Three axial source images (0.5-mm thickness and 0.3-mm interval) were reconstructed with a standard kernel; the data acquisition windows were centered at 70%, 75%, and 80% of the R-R interval. One to 3 data sets with the fewest motion artifacts were loaded onto a 3D work station (Rapidia, Infinitt, Seoul, Korea) for reformation of 5-mm-thick, long-axis curved multiplanar reformation images of the LAD parallel to the septum and short-axis images of 0.5-mm thickness with a gap of 0.9 mm between images (total thickness of 1.4 mm) perpendicular to the long axis of the LAD. Since September 2005, all CTA source images have been stored in a dedicated external hard drive at our institution. Where the reformation plane was not accurate, the specific purpose of the present study was fulfilled by obtaining additional long-axis curved multiplanar reformation and short-axis LAD images.

Image Analysis
All images were analyzed by 2 radiologists who were blinded to the results of CA and reached a consensus. Myocardial bridging was diagnosed when an LAD segment was located within the interventricular gorge and the segment was in contact with left ventricular myocardium, without intervening fat. When the LAD was in contact with the right ventricular wall without contacting the left ventricular myocardium, myocardial bridging was excluded. Myocardial bridg-

Figure 3. Myocardial bridging with full encasement without overlying myocardium in a 45-year-old man with atypical chest pain. A, CA of end-diastolic phase shows minimally narrowed midsegmental lumen (arrows) of the LAD, whereas end-systolic phase depicts a typical dynamic compression (milking effect). Transient occlusion of septal branches is apparent (arrowheads). B, CTA obtained during end-diastolic and end-systolic phase shows narrowing, but motion artifacts limit its accurate quantification. Length also varies according to phase and imaging plane. C, Myocardial bridging seen on a 5-mm-thick long-axis curved multiplanar reformation image of the LAD (left) and on short-axis images obtained perpendicular to the long axis (right). The myocardial bridging segment is fully encased in the interventricular gorge, but there is no overlying muscle (white arrow). In both long- and short-axis images, a septal branch (white arrowhead) is visible, and the right ventricle is outlined by black arrows. Total length of myocardial bridging was 22.4 mm, length of dynamic compression was 18.2 mm, and length of tunneled segment was 0 mm. RV indicates right ventricle; LV, left ventricle; and S, septum.
ing thus diagnosed was assigned to 1 of 2 groups (myocardial bridging with partial encasement and myocardial bridging with full encasement) according to the extent of vessel encasement by the myocardium (Figure 2A). Images were viewed in 2 different window/level settings (110/85 HU and 256/128 HU), and overlying muscle was defined as having similar attenuation behavior to that of left ventricular myocardium (Figure 2B).

In each group, the location, the length of the tunneled segment, and the total length and depth of myocardial bridging were measured and analyzed. With long-axis images, the location of the bridge was classified as proximal, middle, or distal. Where >1 segment was involved, each was listed separately. The dividing line between the proximal and middle segments of the LAD was the first septal branch. The tunneled segment and total length of myocardial bridging were measured on 1.4-mm-thick short-axis images (0.5-mm image thickness with a gap of 0.9 mm between images) by counting the number of images that showed cross-sectional LAD with myocardial bridging; for tunneled segments, LAD with overlying myocardium was measured, and for total length, LAD that was in contact with left ventricular myocardium was measured. The depth of myocardial bridging was determined by perpendicular measurement of the thickness of overlying muscle (>0.7 mm) with use of the short-axis image in which overlying muscle was seen to be thickest.

Conventional Coronary Angiography

All CA was performed by 4 interventional cardiologists according to the standard guidelines of our institution and included a transfemoral or transradial approach and a minimum of 3 different views of the LAD. All images obtained from the 300 patients were stored in a DICOM (Digital Imaging and Communications in Medicine) file on a designated external hard drive, and the set that pertained to each was loaded onto a PACS system (Marosis, Infinitt) for review in random order. One radiologist and 1 cardiologist blinded to the CTA results reviewed images for the presence or absence of dynamic compression in the LAD, reaching a consensus. If a systolic narrowing that was more pronounced than in neighboring normal vessels and was inexplicable in terms of normal flow pattern was observed during the cardiac cycle, dynamic compression was deemed to be present. In patients for whom this was the case, the total length of the dynamic compression was measured during the systolic phase, and vessel diameters were measured during both the end-systolic and end-diastolic phases with an electronic caliper on magnified images. For measurement of the length of dynamic compression at CA, a 5F catheter (outer diameter 1.67 mm) was used as the reference diameter, and the measured values were calculated with adjustment for the magnification factor. The length of the dynamic compression was compared with that of the corresponding tunneled segment measured from short-axis CTA images, and the degree of vessel narrowing was expressed as a percentage. Transient systolic occlusion of septal branches arising from the segment in which dynamic compression was present was observed and recorded. To avoid prejudice of the dynamic compression, the “step-down and step-up” phenomenon, defined as a localized change in the direction of the vessel toward the ventricle, was not used as a diagnostic criterion; this was because observers tend to consider dynamic compression present in patients in whom this phenomenon is observed, especially in equivocal cases.
Data and Statistical Analysis
In descriptive statistical analysis, quantitative variables were expressed as mean±SD and categorical variables as frequencies or percentages. Student’s t test was used to compare quantitative variables such as age, weight, depth, tunneled length, total length, and mean heart rate between patients with and without dynamic compression. The χ² test or Fisher’s exact test was used to compare categorical variables in patients with and without dynamic compression. In addition to probability values, 95% CIs were applied. For all analyses, P<0.05 was considered statistically significant.

In all 39 patients with dynamic compression, the paired t test was used for comparison between the length of the dynamic compression segment by CA and the tunneled length by CT. Pearson correlation analysis was used to determine the relationship between depth, tunneled length, and total length, as assessed by CT, and length of dynamic compression and degree of systolic compression of the tunneled segment, as assessed by CA. All statistical analyses were performed with SPSS version 12.0 (SPSS, Inc, Chicago, Ill).

The authors had full access to and take full responsibility for the manuscript as written.

Results
Eleven patients were excluded because of total occlusion (n=3), bypass surgery (n=3), or artifacts (n=5). Among the remaining 300, CTA revealed that 174 (58%) had myocardial bridging (partial encasement in 57, full encasement in 117); in 40 (13.3%), dynamic compression was observed on CA (1 with partial encasement and 39 with full encasement). The results indicated that dynamic compression occurred almost exclusively in myocardial bridging with full encasement (97.5%; Figure 5). The only dynamic compression observed in myocardial bridging with partial encasement (2.5%) was a short (3.91 mm) systolic narrowing (Figure 4). In 5 patients, there were occlusions or near-occlusions of septal branches at the time of dynamic compression (Figures 3A and 6A).

All cases in which dynamic compression was observed on CA showed myocardial bridging on CTA, and none of the cases with negative findings for myocardial bridging on CTA involved dynamic compression. The 117 patients (39%) with myocardial bridging with full encasement were assigned to 1 of 2 groups (dynamic compression present or absent); they were matched in terms of age, sex, use of β-blocker medication before CTA, heart rate, administration of nitroglycerin during CA, and weight (Table 1).

The total length (21.6±11.2 versus 29.1±15.5 mm; P=0.003) of myocardial bridging, as observed on CTA, correlated with dynamic compression but not with depth (1.2±1.4 versus 1.4±1.0 mm; P=0.283) or tunneled length (5.8±5.8 versus 6.1±6.1 mm; P=0.843). In 3 patients with the greatest depth of myocardial bridging (8.1, 7.01, and 4.23 mm) within the gorge or septum, dynamic compression was not observed, but in 14 (36%) of the 39 patients with myocardial bridging with full encasement and no measurable (>0.7 mm) overlying muscle, dynamic compression was observed (Figure 3). In 1 patient with myocardial bridging with full encasement, dynamic compression was observed in a segment with partial encasement but not in the tunneled segment (Figure 7). Linear correlation analysis revealed a significant relationship between total length of myocardial bridging and length of dynamic compression (Pearson correlation r=0.928, P<0.001). The location of myocardial bridging (inclusion of mid segment) showed a statistically significant correlation with dynamic compression (Table 2).

In all patients (n=39), the length of dynamic compression as measured on CA (22.4±9.5 mm) was considerably greater than that of the corresponding tunneled segment measured on CTA (6.1±6.1 mm; paired t test P<0.001; 95% CI for the difference 13.98 to 18.65), which indicates that dynamic compressions can occur in places other than tunneled segments (Figures 3, 4, and 7). Linear correlation analysis revealed no significant relationship between the percentage of systolic compression (28.55±10.51%) and depth, tunneled length, or total length (Pearson correlation r=0.043, 0.011, and 0.008, and P=0.797, 0.949, and 0.961, respectively).

Discussion
In the present study, the rate of detection of myocardial bridging was 58%, a figure close to the autopsy incidence of 54% to 88%
reported by others. The reasons for the significantly higher detection rate of myocardial bridging by CTA and the higher incidence of dynamic compression revealed by CA (13.3%) than in most other reports are as follows: (1) The inclusion of partial and full encasement on CTA; (2) the use of short-axis images obtained perpendicular to the long axis of the LAD; (3) the consistently high image quality of CTA with 64-section CT; (4) observation of a single artery (LAD) with a specific purpose; and (5) the convenience of our system for reviewers.

Myocardial bridging is generally considered a benign anomaly, but in rare incidences, it is associated with clinical manifestations that have been linked to dynamic compression. Because dynamic compression occurs almost exclusively (97.5% of the time) in myocardial bridging with full encasement, the incidence of myocardial bridging with full encasement (39%) is considered to be more meaningful in the clinical setting.

Dynamic compression has been thought to be caused by the compressing, overlying myocardium of the tunneled segment. However, the current concept cannot explain the following new observations: (1) Dynamic compression occurred in segments without overlying muscle; (2) dynamic compression did not occur in two thirds of patients with overlying muscle; (3) in all patients, the length of the dynamic compression was greater than that of the tunneled segment.

<p>| Table 1. Baseline Characteristics of Patients Who Had Myocardial Bridging With Full Encasement |
|--------------------------------------|--------------------------------------|--------------------------------------|--------------------------------------|</p>
<table>
<thead>
<tr>
<th></th>
<th>Dynamic Compression</th>
<th>No Dynamic Compression</th>
<th>Total</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>39</td>
<td>78</td>
<td>117</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>63.4±10.6</td>
<td>66.2±9.5</td>
<td>65.3±9.9</td>
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<tr>
<td>Male/female ratio</td>
<td>32:7</td>
<td>54:24</td>
<td>86:31</td>
<td>0.139</td>
</tr>
<tr>
<td>β-Blockers (CTA), % (No. of patients)</td>
<td>15.4 (6/39)</td>
<td>21.8 (17/78)</td>
<td>19.7 (23/117)</td>
<td>0.411</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>56.7±6.2</td>
<td>58.3±7.4</td>
<td>57.8±7.1</td>
<td>0.215</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>68.5±9.5</td>
<td>64.6±12.2</td>
<td>65.9±11.5</td>
<td>0.063</td>
</tr>
<tr>
<td>Nitroglycerin (CA), % (No. of patients)</td>
<td>66.7 (26/39)</td>
<td>73.1 (57/78)</td>
<td>70.9 (83/117)</td>
<td>0.472</td>
</tr>
</tbody>
</table>

Unless otherwise specified, data are mean±SD.
segment; (4) total length of the myocardial bridging, rather than depth or length of the tunneled segment, correlated with dynamic compression; and (5) dynamic compression occurred almost exclusively in the LAD.

To confirm the exclusivity of dynamic compression in the LAD, we reviewed the CA of 49 patients (16.3% of subjects in the present study) who had a tunneled segment of the diagonal artery on CTA at the mid segment and found no dynamic compression. This result is in accordance with the observations of others who reported autopsy findings of myocardial bridging of the diagonal and obtuse marginal arteries (incidence of 18% and 40%, respectively); however, angiographically, dynamic compression was observed almost exclusively in the LAD.2

These observations suggest a new hypothesis for the mechanism of dynamic compression, namely, entrapment within the interventricular gorge. We suggest the following conditions for a segment of coronary artery to produce dynamic compression: (1) The vessel is positioned in such a location that a squeezing force can be exerted between the 2 contracting walls (interventricular gorge); (2) a vessel is "trapped" by full muscle encasement, regardless of the depth of the overlying muscle in CTA; and (3) a vessel develops a wall abnormality (endothelial dysfunction) that can affect the degree and pattern of dynamic compression, as well as any pharmacological response.25

The more securely a vessel is entrapped within a confined space (interventricular gorge), the more vulnerable it will be
to the squeezing force between the 2 walls. Provided that the magnitude of the squeezing force and the degree of endothelial dysfunction are not variable, full encasement and a longer total length of myocardial bridging, not the length of the tunneled segment or depth, appear to play an important role in securing the LAD within the interventricular gorge. Although we believe autopsy data demonstrate that the myocardial bridging seen in CTA is likely encased by myocardium of various thicknesses, myocardial bridging with full encasement appears to have sufficient thickness to entrap the LAD securely within the gorge. Prominent septal branches arising from or near the involved segment, which were observed in a number of cases (Figures 3, 4, 6, and 7), may also play a minor role in holding the segment within the gorge, especially in the only case of partial encasement that showed dynamic compression in the present study (Figure 4), although no systematic comparison was made.

Study Limitations
The present study has certain limitations. First, the overlying tissue described in CTA could not be confirmed by autopsy. Second, where the thickness of overlying muscle was <0.7 mm (other studies have used 1.0 mm, whereas the spatial resolution of the CT used in the present study was 0.4 mm), it was difficult to determine the nature of the overlying density (soft tissue), especially where there was motion artifact. Third, initial evaluation of CA for the presence or absence of dynamic compression was based on visual evaluation rather than quantitative analysis. Fourth, the lack of ischemic correlates on stress testing limited the clinical relevance of the findings in the present study. Finally, the present study did not correlate CTA findings with clinical symptoms, the effects of treatment, or follow-up results. Although a considerable number of studies and reports have suggested a possible link between myocardial bridging and clinical manifestations, further studies are needed if the role of CTA in patients with angina-like symptoms or established ischemia but at low risk of coronary artery disease is to be clarified.

Conclusions
The frequency of myocardial bridging observed on CTA was 58%, and CA revealed dynamic compression in 13.3% of all patients studied. Dynamic compression occurred almost exclusively (97.5% of the time) in patients with full encasement of the LAD. To determine the true incidence of myocardial bridging, one that is in accordance with its incidence at autopsy, both groups of myocardial bridging revealed by CTA should be included; nonetheless, the results of the present study suggest that full encasement of the LAD by myocardium, regardless of measurable overlying muscle, is useful for more accurately detecting the type of myocardial bridging that is likely to produce dynamic compression. Entrapment within the interventricular gorge, a new hypothesis for the mechanism of dynamic compression, along with other factors suggested by earlier researchers, appears to play a pivotal role in producing dynamic compression, and this observation may pave the way for further research of myocardial bridging.

Acknowledgment
The authors thank Ki Su Park, RT, the CT technologist, for his invaluable assistance in CT imaging, and Edward Hur, MB, for editorial assistance.

Disclosures
None.

References

**CLINICAL PERSPECTIVE**

Myocardial bridging is clinically significant when associated with regional hemodynamic alterations, and studies have shown that such instances of myocardial bridging are linked to clinical complications that include ischemia, acute coronary syndrome, coronary spasm, arrhythmia, and sudden death, although in the vast majority of cases, myocardial bridging remains clinically silent. The wide discrepancy in the reported prevalence of myocardial bridging between autopsy findings and those of conventional angiography has been attributed to instances of myocardial bridging that do not cause dynamic compression. Recently, CT angiography has been suggested as a tool in the diagnosis of myocardial bridging, with an average depiction rate between that of autopsy and conventional angiography but with a wide variation among authors. In the present study, the frequency of myocardial bridging by CT angiography was 58%, and conventional angiography revealed dynamic compression in 13.3% of all cases. The correlation between CT and conventional angiography revealed that dynamic compression occurred almost exclusively (97.5%) in cases with full encasement of the left anterior descending coronary artery, regardless of the measurable presence of overlying muscle in CT. This finding is useful for more accurately detecting instances of myocardial bridging that are likely to produce dynamic compression. Entrainment within the interventricular gorse, a new hypothesis for the mechanism of dynamic compression, along with other factors suggested by earlier researchers, appears to play a pivotal role in producing dynamic compression, and this observation may pave the way for further research into myocardial bridging.
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