Myocardial Bridging in Adult Patients With Hypertrophic Cardiomyopathy

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OBJECTIVES
This investigation examined the risk of sudden cardiac death and other mortality in adult patients with hypertrophic cardiomyopathy (HCM) who have myocardial bridging diagnosed at coronary angiography.

BACKGROUND
Several reports have associated myocardial bridging with an adverse prognosis in pediatric HCM patients, but the prognosis of myocardial bridging in adult patients with HCM is unknown.

METHODS
The coronary angiograms of 425 patients with HCM (mean age 60 ± 15 years [range 18 to 89 years]) at the Mayo Clinic were examined for the presence of myocardial bridging. Clinical follow-up was conducted to assess mortality. Survival of patients with bridging was compared with HCM patients who also underwent angiography but who did not have evidence of bridging.

RESULTS
A total of 64 patients (15%) had myocardial bridging. The mean follow-up for the entire study was 6.8 ± 5.4 years. There was no difference in survival free of all-cause mortality (5-year estimate, bridging vs. no bridging, 91% vs. 89%; p = 0.60), and sudden cardiac death (95% vs. 97%; p = 0.72). Univariate and multivariate proportional hazards models also did not identify the presence of bridging or specific characteristics of the degree or extent of bridging with a poor outcome.

CONCLUSIONS
This study observed no increased risk of death, including sudden cardiac death, among adult patients with HCM who had myocardial bridging diagnosed at coronary angiography.

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Myocardial bridging occurs when the epicardial coronary arteries are intramyocardial, resulting in systolic compression of a coronary artery on coronary angiography. Myocardial bridging is a well-recognized phenomenon that has a prevalence of 1% to 3% in the general population (1–3). Among patients with hypertrophic cardiomyopathy (HCM), however, this phenomenon is significantly more frequent, with prevalence rates up to 30% (4,5). In studies of pediatric patients with HCM, the presence of myocardial bridging has been associated with the severity of disease, including nuclear perfusion abnormalities, chest pain, ventricular tachycardia, and increased risk of sudden cardiac death (SCD) (6,7). However, there has been no study of the prognostic impact of myocardial bridging in adult patients with HCM. This investigation was therefore undertaken to examine the risk of SCD and other mortality in adult patients with HCM who have myocardial bridging identified at the time of coronary angiography.

METHODS

Patients. Between November 1978 and March 2001, 2,356 patients with HCM were examined at the Mayo Clinic in Rochester, Minnesota. Of these patients, 435 who were ≥18 years of age underwent coronary angiography as part of their cardiac evaluation. Patients whose stored angiographic data were not available (n = 3) or inadequate for review (n = 7) were excluded, leaving a final study population of 425 patients. In all patients, the diagnosis of HCM was based on typical clinical, electrocardiographic, and echocardiographic features, with ventricular myocardial hypertrophy occurring in the absence of any other cardiac or systemic disease, which could have caused the observed hypertrophy (8–10).

Each coronary angiogram was retrospectively examined by an independent observer who had no knowledge of the patients’ medical history or status (i.e., alive or deceased). Myocardial bridging was defined as a change in luminal compression of an epicardial coronary artery of ≥50% during systole (Fig. 1). The cineangiographic projection that demonstrated the greatest degree of bridging was used to determine maximal systolic compression and the total length of the bridged segment. Each artery was examined for the presence of myocardial bridging, maximal percentage of systolic compression, total length of bridging, and reference diameter of the bridged segment. Arterial segment diameters and lengths were determined through calibration with the known diameter of the coronary artery catheter. For patients with multiple sites of bridging, the lengths of each bridged segment were summed to obtain total length. Standard nomenclature of coronary artery anatomy was utilized to define the area affected by bridging (11). The presence of coronary artery disease was defined as luminal...
stenosis of ≥50% in the left main coronary artery or ≥70% in other major epicardial branches (11).

Follow-up evaluation. The Mayo Foundation’s Institutional Review Board approved this study. Clinical follow-up was conducted through mailed questionnaires and/or telephone contact. For deceased patients, circumstances and the primary reason for death were sought through procurement of death certificates and, if possible, medical records and autopsy reports with permission of the next of kin. In all cases, death coding was performed by an investigator blinded to the presence or absence of bridging. Sudden cardiac death was defined as instantaneous and unexpected death with or without documented ventricular fibrillation within 1 h after a witnessed collapse in patients who previously were in a stable clinical condition or nocturnal death with no antecedent history of worsening symptoms. Sudden causes of death that were clearly not organic (e.g., homicide) were not counted as being SCD. Appropriate discharge of an implantable cardioverter-defibrillator (ICD) device for therapy of lethal arrhythmia (i.e., ventricular tachycardia or fibrillation) was considered as SCD for end point analyses. Death due to congestive heart failure was defined as death occurring in the context of long-standing cardiac decompensation, with progression of the disease over the preceding year and development of pulmonary edema or cardiogenic shock.

Statistical analysis. Primary end points were SCD, death due to cardiac causes (including SCD), and all-cause mortality. Follow-up for ≥10 years was censored because the number of patients with bridging who were at risk fell below 15 after this period. For the end point of all-cause mortality, the follow-up time was the interval from the date of the initial evaluation to the time of death or, among survivors, the date of the follow-up evaluation. The date of angiography was considered to be the time of the initial evaluation. The end point of cardiac death included HCM-related death (e.g., SCD), the occurrence of either heart transplantation or death due to stroke. The follow-up time was censored for survivors and patients who died of either noncardiac or undetermined causes. For the end point of SCD, the follow-up time was censored for survivors and patients who died of other causes.

For survival analyses, patients who either had their myocardial bridge resected during surgery (n = 1) or who had concomitant coronary artery disease (n = 97) were excluded (of note, the one patient who had bridge resection also had coronary artery disease). Among this study popu-

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**Figure 1.** Myocardial bridging in a patient with hypertrophic cardiomyopathy. Right anterior oblique views of the left coronary artery during diastole (A) and systole (B). Bridging of the middle left anterior descending coronary artery (LAD) (black arrows on B) is evident during systole. This was associated with complete systolic obliteration of the second septal perforator artery, which is visible only during diastole. There also was mild systolic compression of the distal LAD.

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**Abbreviations and Acronyms**

CI = confidence interval  
HCM = hypertrophic cardiomyopathy  
ICD = implantable cardioverter-defibrillator  
LAD = left anterior descending coronary artery  
SCD = sudden cardiac death  
SM = septal myectomy
involvement of the left anterior descending coronary artery (LAD) with bridging. Other clinical variables considered were severe angina (Canadian Cardiac Society Class III/IV), severe dyspnea (New York Heart Association functional class III/IV), paroxysmal or chronic atrial fibrillation, previous stroke or transient ischemic attack, a family history of sudden death due to HCM, unexplained syncope, maximal wall thickness (<15 mm, 15 to 19 mm, 20 to 24 mm, 25 to 29 mm, ≥30 mm), left ventricular outflow tract obstruction (resting gradient ≥30 mm Hg or provoked gradient ≥50 mm Hg), and septal myectomy (SM).

Because patients with myocardial bridging were considerably younger than patients without myocardial bridging, a second survival comparison was performed. In this comparison, patients who had bridging but who did not have bridge resection or coronary artery disease (n = 54) were age- and gender-matched randomly in a 1:1 fashion to patients with HCM who underwent coronary angiography but who did not have myocardial bridging (i.e., 54 control subjects). Patients with coronary artery disease were not eligible for use as control subjects in this analysis.

The Kaplan-Meier method was used to calculate survival estimates free of the end point events. Survival estimates are reported with a 95% confidence interval (CI). A log-rank test was used to compare survival curves among different patient groups. In an analysis that matched patients with bridging to patients without bridging according to age and gender, Cox models stratified for age and gender were used to examine survival of the two groups. Contingency tables were analyzed for the association with a chi-square value, or, if the patient number in any group was less than 5, the Fisher exact test was used. Comparisons of continuous variables were made with the Wilcoxon rank-sum test. All variables are reported as the mean ± SD. Statistical significance was set a priori at p < 0.05.

RESULTS

Baseline characteristics. Table 1 lists the baseline characteristics of the study population. Myocardial bridging was present in 64 patients (15%). Patients with myocardial bridging were younger and more likely to have a history of SM and family history of HCM compared to patients without bridging (Table 1). A second comparison was performed. In this comparison, patients who had bridging but who did not have bridge resection or coronary artery disease (n = 54) were age- and gender-matched randomly in a 1:1 fashion to patients with HCM who underwent coronary angiography but who did not have myocardial bridging (i.e., 54 control subjects). Patients with coronary artery disease were not eligible for use as control subjects in this analysis.

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Table 1. Baseline Patient Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Bridging Present (n = 64)</th>
<th>No Bridging (n = 361)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>53 ± 14</td>
<td>61 ± 14*</td>
</tr>
<tr>
<td>Male gender</td>
<td>38 (59)</td>
<td>169 (47)</td>
</tr>
<tr>
<td>Dyspnea (NYHA)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class III/IV</td>
<td>30 (47)</td>
<td>182 (50)</td>
</tr>
<tr>
<td>Angina†</td>
<td>23 (36)</td>
<td>125 (35)</td>
</tr>
<tr>
<td>Syncope</td>
<td>9 (14)</td>
<td>47 (13)</td>
</tr>
<tr>
<td>Family history of HCM</td>
<td>14 (22)</td>
<td>4 (13)‡</td>
</tr>
<tr>
<td>Echocardiographic findings</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean MLVWT (mm)</td>
<td>19.5 ± 4.9</td>
<td>20.6 ± 5.1</td>
</tr>
<tr>
<td>LVOT obstruction</td>
<td>30 (47)</td>
<td>180 (50)</td>
</tr>
<tr>
<td>Asymptomatic hypertrophy</td>
<td>32 (50)</td>
<td>154 (43)</td>
</tr>
<tr>
<td>Concentric hypertrophy</td>
<td>17 (27)</td>
<td>129 (36)</td>
</tr>
<tr>
<td>Other hypertrophy</td>
<td>6 (9)</td>
<td>33 (9)</td>
</tr>
</tbody>
</table>

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Follow-up data. Clinical follow-up was achieved in 424 (99.7%) of 425 patients. The mean follow-up duration for the entire study population was 6.8 ± 5.4 years (range 1 month to 24.8 years; median 5.5 years). Ninety-seven patients with either coronary artery disease at the time of angiography and/or bridge resection during follow-up were excluded from survival analyses. Among the remaining 328 patients, there were 58 deaths during the study period. Fifty-one deaths occurred among the 274 patients without bridging: 34 deaths were cardiac in origin, including 10

Table 2. Bridging Segment Characteristics

<table>
<thead>
<tr>
<th></th>
<th>No. vessels with bridging per patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>One</td>
<td>61 (95)</td>
</tr>
<tr>
<td>Two</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Three</td>
<td>2 (3)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Location</th>
<th>No. of bridged segments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Middle LAD</td>
<td>44 (68)</td>
</tr>
<tr>
<td>Middle and distal LAD</td>
<td>11 (17)</td>
</tr>
<tr>
<td>Distal LAD</td>
<td>3 (5)</td>
</tr>
<tr>
<td>Intermediate branch</td>
<td>3 (5)</td>
</tr>
<tr>
<td>Obstructive marginal branch</td>
<td>2 (3)</td>
</tr>
<tr>
<td>Proximal LCx</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Posterior descending artery</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Diagonal branch</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Distal RCA</td>
<td>1 (2)</td>
</tr>
</tbody>
</table>

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Data are presented as the number (%) of subjects or mean value ± SD. LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; RCA = right coronary artery.
SCDs, 14 deaths due to congestive heart failure, 5 due to stroke, 3 due to myocardial infarction, 1 due to heart transplantation, and 1 due to complications related to an out-of-hospital cardiac arrest. Thirteen patients had non-cardiac causes of death. Four patients had undetermined causes. No patient had discharge of an ICD.

Seven deaths occurred among the 54 patients with myocardial bridging without bridge resection or coronary artery disease. Two of these deaths were sudden; two were due to congestive heart failure. Two patients had noncardiac deaths. The cause of death was unknown in one patient.

**Survival analysis. ENTIRE STUDY POPULATION.** Patients with myocardial bridging did not demonstrate a reduction in survival free of any end point, as compared with HCM patients who did not have bridging (Fig. 2). Five-year overall survival was 90.5% (95% CI 83.4% to 99.6%) for patients with bridging and 84.6% (95% CI 79.9% to 89.3%) for HCM patients without bridging (p = 0.42). For the end point of cardiac death, this survival was 93.3% (95% CI 85.9% to 100%) among patients with bridging and 88.9% (95% CI 84.7% to 93.1%) among HCM patients without bridging (p = 0.60). For the end point of SCD, five-year survival was 95.2% (95% CI 94.1% to 100%) for patients with bridging and 96.7% (95% CI 94.2% to 99.1%) among HCM patients without bridging (p = 0.72). Specific characteristics of the degree and extent of bridging were analyzed. Neither the presence of myocardial bridging, total length of the bridged segment, maximal percent diameter reduction, presence of LAD bridging, nor the reference diameter was associated with the study end points in either univariate or multivariate analyses that accounted for difference in age, gender, and other co-variates with statistical association (Table 3).

**AGE- AND GENDER-MATCHED COMPARISON.** In comparison with age- and gender-matched HCM patients without myocardial bridging, HCM patients with bridging did not demonstrate a reduction in survival free of any end point (Fig. 3). Five-year overall survival was 91.5% (95% CI 83.4% to 99.6%) for patients with bridging and 94.3% (95% CI 88% to 100%) for control subjects with HCM who did not have bridging (p = 0.71). For the end point of cardiac death, this survival was 93.3% (95% CI 85.9% to 100%) for patients with bridging and 94.3% (95% CI 88% to 100%) for control subjects with HCM who did not have bridging (p = 0.71). For the end point of SCD, five-year survival was 95.2% (95% CI 94.1% to 100.0%) for patients with bridging and 98.0% (95% CI 88.6% to 100%) for control subjects with HCM who did not have bridging (p = 0.57).

**DISCUSSION**

Myocardial bridging has been associated with the severity of cardiac disease and an increased risk of SCD in pediatric patients with HCM. Because of these reports, there has been confusion about the need for coronary angiography and the appropriate intervention once bridging has been diagnosed in adult HCM patients. The high prevalence of myocardial bridging in HCM patients underscores the need for clarification of the clinical significance of myocardial bridging in adults.
In this investigation, myocardial bridging was not associated with detrimental long-term outcomes, including an increased risk of SCD. To date, the current investigation is the largest reported population of adult patients with HCM who have myocardial bridging. Survival free of the study end points (i.e., overall death, cardiac death, SCD) was identical between patients with and without myocardial bridging. Although patients with bridging were younger than those without bridging, bridging was not predictive after adjustment for age (and male gender), nor was it predictive in a separate analysis with case control for age and male gender. Proportional hazards analysis also did not identify any specific bridging segment characteristics to be predictive of a poor outcome.

There have been a number of reports of unselected patients and patients with HCM that have associated myocardial bridging with cardiac morbidity and risk of death, including SCD. These associations have been explained by reductions in coronary flow from systolic compression, which could be exacerbated during exercise (12). However, there are several confounding issues with regard to the interpretation of these previous studies. Several have been single case reports of HCM patients that, while documenting the co-existence of bridging in a patient, cannot support a causal relation between the presence of bridging and death (13). Further confounding the interpretation of such data is the fact that bridging is more common in patients with myocardial hypertrophy, especially HCM. Patients with HCM have an increased risk of sudden death and cardiac morbidity as compared with the general population. In a study of 23 pediatric patients with HCM, those with myocardial bridging were more likely to have thallium perfusion defects, but multivariate analysis demonstrated that bridging was not predictive of perfusion defects after adjustment for the degree of septal hypertrophy (7). In young
patients, the degree of septal hypertrophy itself has been associated with an increased risk of sudden death (14). In another case series that excluded patients with hypertrophy, myocardial bridging was not associated with ischemia (15).

Several studies have specifically examined the hemodynamic consequences of myocardial bridging and have reported alleviation of cardiac ischemia and symptoms through surgical relief of myocardial bridging (12,16,17). This study did not examine any relationship between bridging and myocardial ischemia. Because the current investigation focused on survival, it does not dispute the possibility that therapy directed at the relief of bridging may be palliative in patients with HCM. However, for the purposes of identifying patients at an increased risk of death, coronary angiography for the detection of bridging and its treatment does not appear to be justified.

**Study limitations.** This was a select group of patients with a high incidence of severe symptoms, which reflects the clinical practice of performing invasive studies in symptomatic patients and the nature of our referral population. Thus, the true incidence of bridging in less symptomatic patients is unknown. Notably, of 29 patients who resided in Olmsted County (i.e., nonreferral population), five (17%) had myocardial bridging. This incidence of myocardial bridging was similar to that of the entire study population (15%). The age discrepancy between patients with and without bridging may raise the possibility of premature death (i.e., selection bias). Further study with prospective screening of patients at a young age would be required to answer this question. Patients with myocardial bridging were more likely to have undergone myectomy than those patients without bridging, and the effect of surgery on improved survival cannot be ruled out, although SM following the initial evaluation was not a predictor of outcomes in this analysis. Another limitation is that there was not a prospective analysis of the occurrence of silent ischemia in these patients.

**Conclusions.** Myocardial bridging is frequent in patients with HCM. This study observed no increased risk of death, including SCD, among patients with HCM who had myocardial bridging, as compared with HCM patients without bridging.

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**References**