Outcome of Repair of Myocardial Bridging at the Time of Septal Myectomy

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Background. **Myocardial bridging describes systolic compression of the muscular investment of a portion of an epicardial coronary artery. We evaluated the outcome of muscular bridge unroofing of the left anterior descending artery at the time of septal myectomy in patients with hypertrophic cardiomyopathy.**

Methods. **We conducted a case-controlled study of 36 patients (23 men; median age, 42 years) with hypertrophic cardiomyopathy and myocardial bridging. Group 1 patients had septal myectomy and concomitant unroofing (n = 13), group 2 patients underwent myectomy alone (n = 10), and group 3 patients were treated medically (n = 13).**

Results. **Angina was more prevalent preoperatively in group 1, 46% compared with 20% in group 2. Preoperative left ventricular outflow tract gradients of 67.8 ± 58.2 mm Hg and 74.1 ± 19.7 mm Hg were reduced to 1.9 ± 2.9 mm Hg in group 1 ($p < 0.0001$) and to 5.6 ± 8.8 mm Hg in group 2 ($p < 0.0001$). In the surgical groups, there were no early deaths or complications related to unroofing. Survival at 10 years was 83.3% in group 1 ($p = 0.297$), 100.0% in group 2, and 67.9% in group 3; there were no late sudden deaths. At follow-up, 77% in group 1 were asymptomatic compared with 70% of patients in group 2 ($p = 0.19$). There was no recurrent angina in group 1.**

Conclusions. **Myocardial unroofing can be performed safely at the time of septal myectomy for left ventricular outflow tract obstruction. Angina was improved, but we found no difference in late survival compared with patients who had myocardial bridging and myectomy alone. Unroofing should be considered in patients with angina who have significant left anterior descending artery bridging and require myectomy.**

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Myocardial bridging describes systolic compression of the muscular investment of a portion of an epicardial coronary artery [1]. Pathologic, morphologic, and radiographic features of myocardial bridging have been well documented in the literature, but little information exists about its clinical significance. Myocardial bridging can be found overlying any coronary artery but occurs most often over the left anterior descending artery (LAD) [2, 3]. Clinical presentations range from no symptoms to angina to myocardial infarction to sudden death [4]. Myocardial bridging is not uncommon in patients with hypertrophic cardiomyopathy (HCM) and has been reported in as many as 15% of patients [5]. The clinical challenge is determining the need for unroofing of the bridged segment at the time of ventricular septal myectomy for obstruction from HCM. To date, there are no recommendations or guidelines regarding the optimal management of myocardial bridging in patients with HCM.

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Patients and Methods

The Mayo Clinic Institutional Review Board approved this retrospective case-control study of 36 patients (23 men) with both HCM and myocardial bridging. Patients were identified from a series of 1,186 patients who had septal myectomy between April 1995 and March 2009. A comparison group of HCM patients with myocardial bridging was selected from a database of our HCM clinic. Groups were matched based on age within 5 years and sex. In group 1 were patients with HCM and myocardial bridging who underwent myectomy and repair of the bridged artery by unroofing (n = 13), in group 2 were patients with HCM and myocardial bridging who had septal myectomy alone (n = 10), and in group 3 were patients with HCM and myocardial bridging who had no surgery and were managed medically (n = 13).

Mean age for all 36 patients was 42 ± 9 years. Preoperative ejection fraction was similar between the two surgical groups (mean, 0.72 ± 0.08 versus 0.74 ± 0.07). Coronary angiography was performed preoperatively on all patients. All available archived angiographic images were reviewed with particular attention to myocardial bridging measurements and coronary artery disease by 2 authors (G.S.S. and D.B.S.). Thickness of a myocardial bridge was defined as very thin, thin, or thick based on reviewer interpretation. Significant myocardial bridging

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was defined as systolic compression of at least 60% and a length of at least 20 mm. The study included review of the Mayo Clinic electronic medical records, our prospective cardiovascular surgical database, and patient surveys obtained at 1, 5, and 10 years after the operation.

Medical management included β-blockers and non-dihydropyridine calcium-channel blockers. Patients were referred for myectomy if their symptoms did not respond to this usual HCM pharmacotherapy or if intraventricular gradients were very large (>100 mm Hg). For the surgical groups, unroofing was offered if the clinical team concluded that the myocardial bridge was contributing to the patient's symptoms. All options (medical management, myectomy alone, and myectomy and unroofing) were offered during the entire study period. Patients' preoperative medications were continued in the postoperative period and were tapered slowly as permitted in the long-term follow-up period. There was no use of antispastic medication in either the unroofing or myectomy alone group.

The vast majority of the operations (90%) were performed by 2 surgeons (H.V.S. and J.A.D.). Access was by means of median sternotomy, and the degree of outflow tract obstruction was documented by direct intracardiac pressure measurements [6] and echocardiography [7]. If the left ventricular outflow tract (LVOT) gradient was low (<30 mm Hg) under anesthesia, provocation was obtained by administering isoproterenol or inducing premature ventricular contractions to determine the maximal gradient [8]. Standard cardiopulmonary bypass with normothermia or mild hypothermia (34°C) was used with aortic occlusion and cardioplegic arrest with cold-blood antegrade cardioplegia. The septum was exposed through an oblique aortotomy, and visualization was facilitated by posterior displacement of the left ventricle with a sponge forceps.

We began the incision in the septum at the nadir of the right aortic sinus and continued leftward toward the mitral valve. Importantly, the area of septal excision should be continued apically beyond the point of mitral-septal contact (usually marked by a fibrous friction lesion referred to as the endocardial scar). The resection was extended apically, and areas of papillary muscle fusion to the septum or ventricular free wall were divided; as well, any anomalous chordal structures and fibrous attachments of the mitral leaflets to the ventricular septum were excised [9, 10].

Unroofing of the myocardial bridge was performed during cardioplegic arrest before or after septal myectomy had been completed. We began by identifying the distal LAD coronary artery, which may be distal toward the apex of the left ventricle. Once normal LAD was identified, we incised the epicardium and dissected proximally into the bridging muscle. There was often additional epicardial fat on top of the myocardial bridge. Dissection was performed with Pott's scissors, and the opened layers of myocardium and epicardial fat were lightly cauterized to control venous bleeding. Importantly, overlying muscle was divided directly over the LAD or with slight deviation toward the left ventricle to reduce the risk of entering the right ventricle (Fig 1). Antegrade cardioplegia was then given, and the LAD was examined to ensure there was no injury to it. After cardiopulmonary bypass, transesophageal echocardiography and needle pressure gradients were again performed to document that the LVOT obstruction had been abolished, that there were no residual intracardiac lesions, and that ventricular function was normal.

Statistical Analysis

Descriptive statistics for categorical variables are reported as frequency and percentage, and continuous variables are reported as mean (± standard deviation) or median (range) as appropriate. Categorical variables were compared among groups using χ^2 test or Fisher's exact test, and continuous variables were compared using Student's t test, analysis of variance, or Kruskal-Wallis test as appropriate. The Kaplan-Meier method was used to draw survival curves and calculate 5-year and 10-year survival statistics. All statistical tests were two-sided with the alpha level set at 0.05 for statistical significance.

Results

All 36 patients had a myocardial bridge over the LAD. All patients were symptomatic, and 56% had multiple symptoms. The most common presenting symptoms in all three groups were dyspnea on exertion (86%), followed by angina (44%). Other symptoms included syncope (11%) and palpitations (5%). In comparing the surgical groups, more patients in group 1 had angina (46% versus group 2, 20%; $p = 0.8$; Table 1).

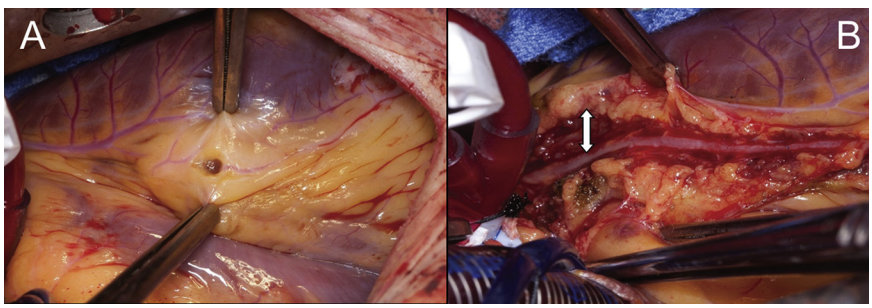

Fig 1. (A) Bridged segment of left anterior descending artery, approximately 40 mm in length. (B) Bridged segment of left anterior descending artery now unroofed. The double arrow shows the thickness of muscle that was overlying the left anterior descending artery.