Midterm results of different treatment methods for myocardial bridging in patients after septal myectomy

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Abstract

Background: Myocardial bridging (MB) is commonly treated in patients with hypertrophic cardiomyopathy (HCM). However, whether and how MB should be treated in patients with hypertrophic obstructive cardiomyopathy (HOCM) who underwent septal myectomy remain unclear. Methods: A total of 823 adults with HOCM who underwent septal myectomy at the Fuwai Hospital from 2011 to 2017 were retrospectively studied. Results: Overall, 31 events occurred: 24 patients died and 7 had nonfatal myocardial infarction (MI). The 3-year cumulative event-free survival of all-cause death (97.9% vs 100% vs 100% vs 98.4%, p=0.89) and cardiovascular death (98.3% vs 100% vs 100% vs 98.4%, p=0.63) were similar among the four groups (non-MB, CABG, unroofing, untreated, respectively). The 3-year cumulative event-free survival of nonfatal MI (100% vs 97.5% vs 98.0% vs 89.9%, p<0.001) and combined endpoints (97.9% vs 97.5% vs 98.0% vs 88.4%, p=0.02) were significantly lowest in untreated MB. Cox regression analysis indicated that untreated MB was a significant independent predictor of combined endpoints (hazard ratio [HR]: 4.06, 95% confidence interval [CI]: 1.60–10.32, p<0.001). Moreover, 49 patients underwent coronary artery computed tomography after surgery. The patency rate of the saphenous vein graft (SVG) was significantly higher than that of the left internal mammary artery (LIMA) (13.3% vs 84.2%, p<0.001). No MB was detected in the unroofing group. Conclusions: Surgical MB treatment could be beneficial and performed safely during septal myectomy. Myocardial unroofing is the recommended treatment for MB, and unroofing when technically possible may be preferable for long-term outcomes.

Introduction

Myocardial bridging (MB) occurs when a band of cardiac muscle overlies the intramural segment of the coronary artery, resulting in systolic compression that is observable on coronary angiography.[1] MB is a well-recognized phenomenon that has 1–3% prevalence in the general population. Its clinical presentation ranges from no symptoms to chest pain, myocardial infarction (MI), and even sudden death.[2] MB is more notably prevalent among patients with hypertrophic cardiomyopathy (HCM), with a prevalence of up to 30%.[3] Previous studies have reported that in pediatric patients with HCM, the presence of MB is associated with disease severity.[4,5] However, for patients with hypertrophic obstructive cardiomyopathy (HOCM) requiring surgery, whether and how MB should be treated remain unclear.

To date, there are no recommendations or guidelines regarding the optimal management of MB in patients with HOCM. The existing surgical treatments of MB mainly include coronary artery bypass grafting (CABG) and unroofing, which involves the use of a saphenous vein graft (SVG) and the left internal mammary artery (LIMA).[6] However, it has not been established which between CABG and unroofing is better. Therefore, in

this study, we evaluated the midterm outcomes of these different treatment methods in patients with HOCM complicated with MB.

Materials and Methods

Patients

We included 823 consecutive patients with HOCM evaluated at the Fuwai Hospital in Beijing between 2011 and 2017. All patients underwent coronary arteriography. MB was defined as an epicardial coronary artery systolic compression of [?]50% of the diameter on coronary arteriography (a representative myocardial bridge is shown in Figure 1). In this study, the MB group was defined as patients with HOCM complicated with MB and the non-MB group as HOCM patients without MB. The diagnostic criteria and surgical indications of HOCM were consistent with the 2011 American Heart Association/American College of Cardiology (AHA/ACC) guidelines, which mainly include unexplained septal hypertrophy with a thickness of >15 mm.[7] The indications for septal myectomy were severe symptoms or syncope or near-syncope despite optimal medical therapy and an LV outflow tract (LVOT) gradient >50 mmHg at rest or with provocation. We excluded patients with coronary atherosclerosis heart disease and those younger than 18 years.

The study was approved by the Ethics Committee of Fuwai Hospital, Chinese Academy of Medical Sciences. Informed consent was obtained from all patients, in accordance with the principles of the Declaration of Helsinki.

Cardiac surgery

The septal myectomy procedure has been described in detail in our previous publication.[8] Indications for MB intervention mainly include chest pain and the degree and length of compression of MB. In addition, the operative procedure is mainly determined by the degree and length of compression. For patients with relatively shallow and short MB, myocardial unroofing was performed. However, for those with deep and extensive MB, CABG was conducted. Moreover, MB was not treated for patients with no symptoms of chest pain and with shallow and short MB. After septal myectomy and aortic incision suturing, we performed myocardial unroofing under the circumstance of cardiac arrest. First, we determined the position of the MB and then cut the epicardium right above the coronary artery and maintained its integrity (Figure 2). After this unroofing, 6-0 Prolene was used to continuously suture the adipose tissue on the surface of the heart. CABG was performed according to previous studies, and other related operations performed according to preoperative evaluation and intraoperative exploration. Two grafts, namely, LIMA and SVG, were used in all CABG procedures.

Endpoints and follow-up data

The clinical status of the participants was ascertained through telephone interviews at least once a year after septal myectomy. Those patients who died were treated as the endpoints, and the follow-up time was defined as their time of death. The last follow-up of the survivors was conducted in June 2018. The clinical endpoints in this study were defined as all-cause death, cardiovascular death, and nonfatal MI. The combined endpoints include the former three endpoints. Cardiovascular death was defined as any death with no clear non-cardiac cause. MI was diagnosed according to the standard definition (serum cardiac biomarker elevation with symptoms of ischemia and/or ECG changes indicative of new ischemia/infarction).

Echocardiography

Echocardiographic examinations were performed by one experienced physician. The diameter of the cardiac chambers was expressed as the maximum value of the anteroposterior diameter in cardiac cycles. The diameter of the ascending aorta was approximately 4 cm greater than that of the aortic valve during diastole. The thicknesses of the interventricular septum (IVS) and ventricular wall were determined during diastole. Aside from the maximum thickness, the representative thickness of the IVS, which is usually the thickness of the point 25 mm under the right coronary sinus nadir, was also recorded to indicate the overall thickness. The LVOT gradient was calculated using the simplified Bernoulli equation. The ventricular ejection fraction

measurements were determined according to the American Society of Echocardiography recommendations. These methods have been provided in greater detail in our previous publication.[8]

Statistical analysis

The results are expressed as mean ± standard deviation or percentage, as appropriate. Student t-test or Mann-Whitney U test (used when data does not conform to a normal distribution) were used for independent samples, paired t-test or the Wilcoxon signed-rank test (used when data does not conform to a normal distribution) for paired data, and Mann-Whitney U test were used to compare continuous variables. Furthermore, χ^2 or Fisher's exact tests were used to compare nominal variables, as appropriate; Fisher's exact test is used if one of the expected frequencies is less than 5, and the χ^2 test is used if each frequency is greater than 5. Differences among the different groups were compared using one-way analysis of variance (differences between groups of 3 or more were examined). The Kaplan–Meier method was used to calculate survival free from the endpoint events. A log-rank test was used to compare survival curves among the different patient groups. Univariable and multivariable Cox regression analyses were used to assess the association of individual variables with combined endpoints. Age, sex, and variables with p <0.1 on univariate analysis were incorporated into a multivariate analysis. All the reported probability values were two-tailed, and a p-value <0.05 was considered statistically significant. SPSS version 24.0 Statistical software (IBM) and GraphPad Prism 7.0 (GraphPad Software Inc., La Jolla, CA, USA) were used for calculations and illustrations, respectively.

Results

Baseline patient characteristics

A total of 823 consecutive patients with HCM were included in this study (500 male patients, 60.8%; mean age, 45.2±13.8 years). In the cohort, 203 patients (24.7%) had MB, and 620 (75.3%) patients had no MB. The baseline characteristics of the entire population and subgroups based on the presence or absence of MB are described in Table 1. The non-MB group contained more females and the patients were older. The MB group had a higher prevalence of chest pain than the non-MB group (23.4% vs 36.9%, p<0.001). Detailed information is presented in Table 1.

Perioperative data among the different groups

Patients with MB were divided into three groups, namely, CABG group (n=90), myocardial unroofing group (n=52), and untreated group (n=61). The perioperative period was defined as within 30 days after the operation. Compared with other groups, the myocardial unroofing group had a longer cardiopulmonary bypass time and aortic cross-clamping time. In addition, the degree of arterial compression was higher in the myocardial unroofing group than in the CABG and untreated groups. However, there was no difference between the CABG and untreated groups. The length of MB was longer in the CABG group than in the unroofing and untreated groups, and 3 patients died of heart failure during the perioperative period. There was no difference between the unroofing and untreated groups. The postoperative hospital stays and postoperative ventilation time did not differ among the different groups. Most importantly, no difference was found between the three groups regarding perioperative death and the concomitant procedure (Table 2).

Follow-up

The New York Heart Association (NYHA) class, percentage of systolic anterior motion (SAM), and LVOTO were significantly improved after the surgery. There were no differences in the incidences of LVOTO and SAM among the four groups. Detailed information is presented in Table 3. In addition, coronary artery computed tomography was performed in 49 patients 1 year after the surgery (15 in the CABG with LIMA, 19 in the CABG with SVG group, and 15 in the myocardial unroofing group). The graft remained patent in only 2 (13.3%) patients in the LIMA group and 16 (84.2%) patients in the SVG group. However, no MB was detected in the myocardial unroofing group.

The mean follow-up period was 34.2±17.2 months. A total of 31 events occurred: 24 patients died (20 patients

without MB, 1 patient in the CABG group, 1 patient in the unroofing group, and 2 patients in the untreated group) and 7 patients (2 patients in the CABG group, 1 patient in the unroofing group, and 4 patients in the untreated group) had a non-fatal MI. Of the aforementioned deaths, 4 were due to cancer, 10 due to sudden cardiac death, 8 due to heart failure, 1 due to MI, and 1 due to infective endocarditis. The 3-year cumulative event-free survival of all-cause death (p=0.89) and cardiovascular death (p=0.63) were similar among the four groups (Figure 3 A and B). However, compared with the other three groups, the 3-year cumulative event-free survival of nonfatal MI (p<0.001) and combined endpoints of all 3 events (including all-cause death, cardiovascular death, and nonfatal MI) (p=0.02) were significantly lower in the no-treatment MB group (Figure 3 C and D).

Univariable and multivariable Cox regression analysis were performed to investigate the predictors of combined endpoints in the follow-up period (Table 4). Cox regression analysis indicated that non-treated MB was a significant independent predictor of combined endpoints (HR: 4.06, 95% CI: 1.60–10.32, p<0.001). In addition, the NYHA class III or IV (HR: 4.72, 95% CI: 1.12–19.92, p=0.04) and postoperative hospital stays (HR: 1.07, 95% CI: 1.03–1.11, p=0.001) were also independent predictors of combined endpoints of all three events.

Discussion

In this study, the prevalence of MB in patients with HCM was 24.7%. The main findings are as follows. First, in patients with HOCM, the surgical treatment of MB can significantly reduce the incidence of nonfatal MI and combined endpoints (including all-cause death and nonfatal MI). Second, considering the patency rate of the graft vessels, myocardial unroofing is the recommended treatment for eligible patients, and every effort to perform unroofing when technically possible may be preferable for long-term outcomes.

The prognostic implications of MB in patients with HCM are controversial. It has been suggested that compared with non-MB patients, patients with MB have more abnormalities during exercise testing and a greater incidence of chest pain, cardiac arrest, and ventricular tachycardia.[9] In one study involving pediatric patients with HCM, the presence of MB may be an additional risk factor for sudden cardiac death and myocardial ischemia.[4] In contrast, another study found that MB does not result in myocardial ischemia and may not cause arrhythmias or sudden death in children with HCM.[5] In addition, a previous study reported that MB is associated with a higher prevalence of chest pain. However, the chest pain assessment in patients with HOCM is complicated because chest pain may be related to an underlying cardiomyopathy, associated fixed coronary artery disease, or MB and its sequelae.[10] In our study, all patients underwent coronary arteriography, excluding patients with coronary heart disease, and we found that the MB group had a higher prevalence of chest pain before the surgery than the non-MB group.

In this study, we found that the incidences of nonfatal MI and combined endpoints were significantly higher in patients with untreated MB after surgery, but there was no difference between the non-MB and the treated MB groups after surgery. Some case reports have shown that surgical MB treatment can significantly improve symptoms and decrease the risk of adverse cardiovascular events in patients with HOCM.[11-14] Therefore, we believe that the surgical treatment of a myocardial bridge may be beneficial and can be performed safely during septal myectomy.

Presently, there are two main surgical procedures for the treatment of MB: myocardial unroofing and CABG. However, it has not been established which of these two methods is better. A previous study compared the results of CABG and myocardial unroofing in isolated MB and suggested that patients who are refractory to medication should actively undergo surgical procedures, such as myocardial unroofing or CABG, while myocardial unroofing should be recommended as the first option because of its safety and satisfactory results.[15,16] In this study, we found that myocardial unroofing was better than CABG in terms of chest pain relief and a higher occlusion rate in the CABG group. Our results are consistent with those of previous studies suggesting that surgical unroofing in carefully selected patients with MB can be performed safely as an independent procedure with significant postoperative improvement in symptoms.[17-19] In addition, we found that the myocardial unroofing group had a higher degree of arterial compression and the length of

MB was longer in the CABG group. In fact, longer and deeper MB might be associated with a higher risk of ventricular rupture, bleeding, and aneurysm formation as a result of unroofing.[20] In this study, we found that during the relatively long follow-up period, SVG had a higher primary patency than LIMA. Our results are consistent with the results of a previous study that demonstrated that LIMA patency in an isolated MB of the left anterior descending artery was low, and that SVGs should be considered in cases of CABG for MB.[21] Multiple studies have reported graft dysfunction and occlusion in cases of competitive flow. [21,22]Low-grade narrowing of the LAD that results in higher competitive flow, low-grade stenosis of a bypassed coronary artery, the muscular layer of LIMA, and the potential for competitive flow of MB contributed to the occlusion in those patients who underwent CABG using LIMA. In addition, it is known that during diastole there is almost normal coronary blood flow with a high probability of competitive blood flow through the graft. This situation, together with the high sensitivity of the LIMA to competitive coronary flow, might explain the remarkably low patency of LIMA grafts.

From our experience, in clinical practice, myocardial unroofing is the recommended treatment for eligible patients with HOCM complicated with MB, and every effort to perform unroofing when technically possible may be preferable for long-term outcomes. Owing to the higher risk of ventricular rupture, bleeding, and aneurysm formation as a result of unroofing, septal myectomy and myocardial unroofing should be performed by expert cardiac surgeons who are experienced in both operations.

There were some limitations to our study. First, this was a retrospective study, and at different instances, there were differences in the treatment of HOCM complicated with MB. Because of our understanding of the disease and the growing maturity of our surgical techniques, we used different methods for treating MB at different times. In addition, Second, few patients underwent coronary artery computed tomography or coronary angiography after surgery. Hence, we could not accurately evaluate the results of unroofing and the primary patency of the bridge after surgery. Third, short period was considered for observing cardiac mortality and morbidity. In the future, a long-term follow-up for these patients is needed to obtain a better understanding of the results of the different treatment methods for MB in patients with septal myectomy. Fourth, it is an inherent limitation of this observational study that the comparison is uncontrolled for selection bias, and the decision on the intervention might be affected by the baseline characteristics. However, it should be noted that the general differences among the four groups were very mild and the differences in outcomes were significant. In addition, the small number of events and uncontrolled nature were also major limitations of our study. Finally, patients with HOCM who underwent septal myectomy are known to have a better prognosis, which is close to that of an age- and sex-matched general population. Therefore, we had to admit that the number of events is small in our study.

Conclusions

Surgical MB treatment is beneficial and can be performed safely during septal myectomy. Because there is a risk of graft vessel blockage in the long-term, myocardial unroofing is the recommended treatment for MB, and every effort to perform unroofing when technically possible may be preferable to achieve long-term outcomes.

Figure Legends

Figure 1 Representative myocardial bridging shown in coronary angiography.

Figure 2 Intraoperative diagram

After cut the epicardium right above the coronary artery and kept its integrity.

Figure 3 Kaplan-Meier curves for the endpoint.

(A) All cause death (B) cardiovascular death (C) nonfatal myocardial infarction and (D) the combined endpoints.

Table 1 Baseline patients' characteristics

Variable	Total (n=823)	non-MB (n=620)	With MB (n=203)	p-value
Age, years	45.2±13.8	46.3±13.3	41.9±14.8	<0.001
Male gender	500 (60.8%)	359 (57.9%)	141 (69.5%)	0.003
Body mass index, kg/m^2	24.9±3.6	25.1±3.6	24.4±3.8	0.02
Family history of HCM	127 (15.4%)	85 (13.7%)	42 (20.7%)	0.02
Atrial fibrillation	83 (10.1%)	67 (10.8%)	16 (7.9%)	0.23
Pulmonary hypertension	63 (7.7%)	46 (7.4%)	17 (8.4%)	0.66
NYHA class III or IV	666 (80.9%)	506 (81.6%)	160 (78.8%)	0.38
Comorbidities				
Hypertension	160 (19.4%)	133 (21.5%)	27 (13.3%)	0.01
Hyperlipemia	104 (12.6%)	87 (14.0%)	17 (8.4%)	0.04
Diabetes mellitus	43 (5.2%)	32 (5.2%)	11 (5.4%)	089
Clinical presentation				
Dyspnea	280 (34.0%)	219 (35.3%)	61 (30.0%)	017
Chest pain	220 (26.7%)	145 (23.4%)	75 (36.9%)	<0.001
Palpitation	103 (12.5%)	89 (14.4%)	14 (6.9%)	0.005
Amaurosis	71 (8.6%)	53 (8.5%)	18 (8.9%)	0.89
Echocardiographic indices				
Aorta, mm	30.3±4.6	30.4±4.6	30.1±4.5	0.42
Left atrium, mm	44.3±7.7	44.5±7.7	43.8±7.8	0.31
LVEDD, mm	41.9±5.4	42.1±5.4	41.7±5.4	0.43
Max IVST, mm	22.8±5.4	22.8±5.5	22.8±5.2	0.07
Posterior LV wall, mm	12.0±2.7	12.1±2.7	11.7±2.8	0.96
LVOT gradient, mm	80.9±27.2	81.9±27.8	78.4±25.3	0.13
LVEF, %	71.5±6.3	71.5±6.4	71.6±6.1	0.83
Medical therapy				
Beta-blockers	743 (90.3%)	560 (90.3%)	183 (94.2%)	0.44
Calcium-channel blockers	106 (12.9%)	89 (14.4%)	17 (8.4%)	0.03

Values are presented as percentage, mean ± SD.

NYHA= New York Heart Association; IVST=interventricular septal thickness; LVEDD=left ventricular end diastolic diameter; LVOT =left ventricular outflow tract gradient; LVEF=left ventricular ejection fraction.

Table 2 Perioperative data among the different groups

Variable	non-MB (n=620)	CABG (n=90)	Unroofing (n=52)	Untreated (n=61)	P value
Degree of MB compression, %	-	78.5±9.8*	85.8±8.1	75.4±12.4$^{*,\#}$	<0.001

Variable	non-MB (n=620)	CABG (n=90)	Unroofing (n=52)	Untreated (n=61)	P value
Length of MB, mm	-	28.9±15.2*	22.5±15.6	16.8±10.9*,$	<0.001
Concomitant procedures					
MVP	63 (10.2%)	5 (5.6%)	5 (9.6%)	9 (14.8%)	0.32
MVR	12 (1.9%)	2 (2.2%)	2 (3.8%)	0 (0%)	0.36
DVR	3 (0.5%)	0 (0%)	0 (0%)	0 (0%)	0.64
TVP	59 (9.5%)	4 (4.4%)	1 (1.9%)	6 (9.8%)	0.06
RVOTO correction	5 (0.8%)	1 (1.1%)	2 (3.8%)	1 (1.6%)	0.41
WPW pathway amputation	7 (1.1%)	0 (0%)	0 (0%)	1 (1.6%)	0.37
Maze procedure	36 (5.8%)	4 (4.4%)	1 (1.9%)	4 (6.6%)	0.55
Perioperative pacemaker	12 (1.9%)	1 (1.1%)	0 (0%)	1 (1.6%)	0.53
Cardiopulmonary bypass time, min	103.9±41.8	108.1±52.2	122.9±39.5	114.0±49.0	0.009
Aortic cross-clamping time, min	69.1±28.8	77.9±49.7*	84.8±21.5	72.1±33.4*	0.001
Postoperative ventilation time, h	19.7±20.7	21.1±24.9	20.4±12.3	22.6±18.2	0.71
Postoperative hospital stays, d	8.5±4.6	9.6±5.3	7.6±2.0	8.5±3.5	0.05
Perioperative death	2 (0.32%)	0 (0%)	0 (0%)	0 (0%)	0.636

*p<0.05 when compared with the unroofing group.

#p>0.05 when compared with the CABG group.

$p>0.05 when compared with the unroofing group.

Values are presented as percentages, mean ± SD.

MVP=mitral valvuloplasty; MVR=mitral valve replacement; DVR=double valve replacement; TVP= Tricuspid valvuloplasty; RVOTO=right ventricular outflow tract obstruction; CABG=coronary artery bypass graft.

Table 3 Clinical and echocardiographic data at the last-follow-up visit

Variables	non-MB (n=620)	CABG (n=90)	Unroofing (n=52)	Untreated (n=61)	P value
NYHA class					
Baseline	2.9±0.5	2.9±0.6	2.8±0.4	2.9±0.3	0.29

Variables	non-MB (n=620)	CABG (n=90)	Unroofing (n=52)	Untreated (n=61)	P value
Last follow-up Max LVOT gradient >30mmHg	1.1±0.4	1.2±0.5	1.0±0.1	1.1±0.3	0.14
Baseline	620 (100%)	90 (100%)	52 (100%)	61 (100%)	1.00
Last follow-up	38 (6.1%)	4 (4.4%)	4 (7.7%)	6 (9.8%)	0.57
SAM					
Baseline	449 (72.4%)	64 (71.1%)	41 (78.8%)	47 (77.0%)	0.64
Last follow-up	1 (1.1%)	0 (0%)	2 (3.3%)	13 (1.9%)	0.39
Max IVST, mm					
Baseline	22.8±5.5	23.4±5.2	23.6±5.8	21.3±4.3	0.07
Last follow-up	15.1±4.5	16.1±4.5	15.2±3.8	14.4±2.8	0.13

MB=myocardial bridging; IVST=interventricular septal thickness; LVOT =left ventricular outflow tract; LVWT=left ventricular wall thickness.

Table 4 Univariable and Multivariate Cox Regression Analyses to Predicts combined endpoints

Variables	Univariable	Univariable	Multivariable	Multivariable
	HR (95% CI)	p	HR (95% CI)	p
Age	1.008 (0.98-1.04)	0.60		
Male	1.10 (0.53-18.56)	0.80		
NYHA class III or IV	4.42 (1.05-18.56)	0.04	4.72 (1.12-19.92)	0.04
Chest distress	0.48 (0.23-0.99)	0.05		
LVEF	0.95 (0.90-1.00)	0.04		
Beta-blockers	0.33 (0.14-0.82)	0.02		
Maze procedure	3.26 (1.25-8.51)	0.02		
Postoperative hospital stays	1.07 (1.03-1.11)	0.001	1.07 (1.03-1.11)	0.001
Reference		0.035		0.03
CABG	1.35 (0.39-4.68)	0.64	1.29 (0.37-4.50)	0.69
Myocardial unroofing	1.66 (0.38-7.24)	0.50	2.12 (0.48-9.39)	0.32
Untreated	3.97 (1.57-10.02)	0.004	4.06 (1.60-10.32)	0.003

LVEF=left ventricular ejection fraction; CABG=coronary artery bypass graft;

Reference=patients without myocardial bridging.

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