

Myocardial Bridge: Surgical Outcome and Mid-term Follow up

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Abstract

Background- Myocardial bridge consists of muscle fiber bundles lining an epicardial coronary artery for a variable distance. Although myocardial bridge is associated with a benign prognosis, its presence has also been considered a cause of angina, myocardial infarction, malignant arrhythmia and sudden death. There is no general consensus about therapeutic strategies in symptomatic patients with myocardial bridge (medical therapy, coronary artery bypass surgery, coronary stenting, supra-arterial myotomy). We report results of surgery and long-term follow up in 26 patients who had disabling symptoms due to myocardial bridge refractory to medical therapy.

Methods- From 1999 to 2004, among more than 18,800 coronary angiographies which were performed in our department, 290 (1.5%) cases had the angiographic diagnosis of myocardial bridge. From these, 26 (9%) patients underwent surgical myotomy for treatment of myocardial bridge causing significant systolic arterial compression. The patients (19 male, 7 female) had a history of typical chest pain and positive exercise test. All of them were examined with radionuclide study preceding angiography, which was positive for ischemia in 20 cases (76%). Coronary angiography and left heart catheterization revealed impaired blood flow due to myocardial bridge in left anterior descending artery in all patients and there was additional atherosclerotic stenosis of coronary arteries in 6 and mitral valve disease in one patient. Supra arterial myotomy was performed in all patients.

Results- There was no mortality or major intraoperative complication. Postoperative scintigraphic and angiographic studies demonstrated restoration of coronary blood flow and myocardial perfusion without significant residual compression of the artery, except in one patient who had recurrent anginal chest pain after operation and coronary angiography showed residual narrowing in the LAD despite myotomy. This patient underwent CABG of LIMA to distal LAD. During 7-81 months of follow-up (mean: 34.2 ± 21), only two patients had symptoms of angina which did not show significant residual compression, and symptoms were controlled by medical treatment.

Conclusion- In conclusion, surgical relief of myocardial ischemia due to myocardial bridge can be accomplished with very low operative risk and excellent mid term results (*Iranian Heart Journal 2007; 8 (2): 39-43*).

Key words: myocardial bridge; supra arterial myotomy; coronary artery bypass surgery ■ coronary angiography

Muscle overlying the intramyocardial artery, first mentioned by Reyman in 1773,¹ is segment of an epicardial coronary known as myocardial bridge (MB).

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It is characterized by systolic compression of the tunneled segment, which remains clinically silent in the vast majority of cases. The angiographic prevalence of bridging has been reported as 0.5 -1.6 percent.²⁻⁴ A high prevalence has been reported in heart transplant recipients and in patients with hypertrophic cardiomyopathy.³ A milking effect or transient narrowing of bridged artery during systole can cause a wide variety of symptoms including typical angina, myocardial infarction, malignant arrhythmia and sudden cardiac death. In the literature many case reports have described a variety of symptoms attributed to bridging and prompt relief of symptoms following the treatment of myocardial bridges. In this study, we report the results of surgery and mid-term outcome in a series of 26 patients with symptomatic myocardial bridging who underwent supra-arterial myotomy.

Methods

Between 1997 and 2004, among 18,800 coronary angiographies which were performed in our center, we detected 290 cases (1.5%) with an angiographic diagnosis of myocardial bridge. Among them, 26 (9%) patients were referred for surgery due to disabling symptoms or resistance to medical therapy.

These patients were symptomatic: 21 (80%) had chest pain, 4 (15%) had exertional dyspnea, and one case suffered from early fatigue. There was a positive history of acute coronary syndrome in 12 patients (46%), and one patient had recurrent attacks of severe chest pain and admission to CCU and another one presented with acute anterior myocardial infarction. Demographic characteristics and coronary risk factors are shown in Table I.

Table I. Patient characteristics

AGE (years)	48±5
MALE	73%
Chest pain	80%
ACS	46%
LVH	30%
EKG change	76%
ETT (positive)	92%
MPI (positive)	76%
HTN	33%
SMOKING	30%
CABG	30%
Systolic compression	70 ± 8%

ACS: Acute Coronary Syndrome, **LVH:** Left ventricular hypertrophy, **ETT:** Exercise tolerance test, **MPI:** Myocardial perfusion imaging, **HTN:** Hypertension, **CABG:** Coronary artery bypass graft

Myocardial bridges were localized in the middle portion of LAD and were diagnosed by systolic compression and angiographic milking. The diameter of the tunneled segment was measured by digital caliper in systole and diastole. The systolic reduction of intraluminal diameter was between 60 – 80% (average 70 ± 8%, Fig. 1).

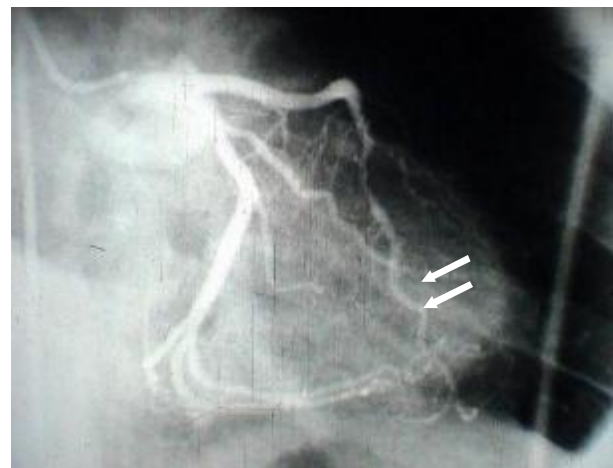


Fig. 1. Coronary angiography shows myocardial bridge of left anterior descending (LAD) artery with 80% systolic compression before surgery.

Resting ECG showed ischemic ST – T changes in 11 cases and LVH in 9 cases (34%). Transthoracic echocardiography finding in these patients was consistent with concentric LVH in 8 patients, although none of them proved to be hypertrophic cardiomyopathy.

Exercise test under the Bruce protocol was performed in all the patients and was positive in 24 patients (92%). Myocardial perfusion scan with Thallium 201 also was performed in all the patients, which showed reversible myocardial perfusion abnormality in anterior wall and septum in 20 cases (76%). Concomitant atherosclerotic lesions were present in 6 cases, and one patient had coronary artery dissection in LAD just proximal to the myocardial bridge.

Results

Unroofing of the myocardial bridge was performed through median sternotomy. The surgical method in the first three patients was without cardiopulmonary bypass (off-pump, beating heart). However, this technique was changed to cardiopulmonary bypass because of LAD traumatization in the third patient.

In 5 patients, saphenous vein grafts were used to bypass diseased segments of atherosclerotic arteries other than LAD. In addition, 2 patients had LIMA to LAD, one of them due to LAD significant atherosclerotic lesion and the other presented with acute anterior MI and was shown to have coronary artery dissection at the proximal portion of LAD. In one patient, open mitral valve commissurotomy was performed because of severe mitral stenosis. Myotomy site was sutured in continuous locked at both sides to prevent epicardial venous bleeding.

There was no perioperative mortality and through the follow-up period. One patient had recurrent anginal chest pain after operation, and coronary angiography revealed residual narrowing of LAD despite myotomy.

This patient underwent CABG by grafting LIMA to the distal of LAD.

Perioperative myocardial infarction was not observed by serial measurement of CK-MB and serial ECGs.

All the patients were followed between 7-81 months (average 34 ± 2 months). During this period, there was no mortality. Two cases had recurrence of chest pain, and the remainder were symptom-free and all the cases were free of major cardiac events. Exercise test and myocardial perfusion scan were performed 6-12 months following operation and did not show any residual ischemia.

Twelve patients underwent postoperative coronary angiography for an evaluation of residual narrowing, which showed a complete resolution of the systolic squeezing of the vessel (Fig. 2).

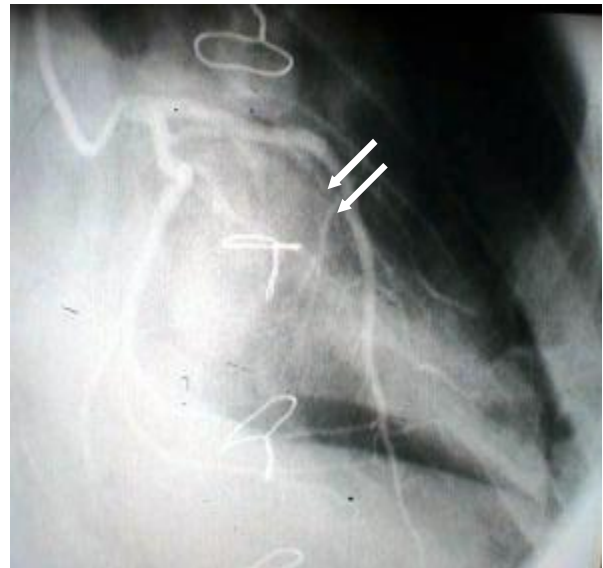


Fig. 2. Coronary angiography of the same patient as in Fig.1 after supra-arterial myotomy shows no systolic compression.

In those two cases who were symptomatic, no apparent systolic compression was seen and symptoms were alleviated by prescribing beta blockers.

Discussion

Myocardial bridges are relatively common findings. Their prevalence differs on the basis of study method, with a much higher rate at autopsy (50%)⁴ versus angiography (0.5-1.6%). Although being asymptomatic in many cases, and found only by chance, their presence has also been considered a cause of angina, malignant arrhythmia, myocardial infarction and sudden death.^{5,6}

They are diagnosed *in vivo* by angiographic milking and systolic compression of coronary arteries that disappear during diastole. Through new imaging techniques like IVUS (intravascular ultrasound) and intracoronary Doppler ultrasound (ICD) more has been known about the mechanism of ischemia and morphological and functional features of myocardial bridging.⁷

In the literature, there are different treatment options to improve quality of life in symptomatic patients, although hard evidence for a favorable effect on mortality and morbidity is missing.⁸ Three treatment strategies have been evaluated: 1) medical treatment with calcium blockers and beta blockers, 2) surgical myotomy and/or CABG, and 3) stenting of the tunneled segment.

In a recent provocative report of a relationship between sudden death and the presence of myocardial bridging in children with familial HCM, Yetman and associates suggested that surgical unroofing of the coronary artery can prevent sudden death.⁹ Sorajja et al. observed no increased risk of death, including sudden cardiac death, among adult patients with HCM who had myocardial bridge.¹⁰

In subjects refractory to medication, surgical myotomy, first reported by Binet et al. in 1975, abolishes clinical symptoms and is associated with reversal of local myocardial ischemia and an increase in coronary flow.^{11,12}

In 1995, Stables et al.¹³ first reported coronary stenting as an interventional approach to severe myocardial bridging refractory to medication with successful short-term results. However, because about 50% of these

patients develop restenosis and major periprocedural complications, coronary intervention is not a generally recommended approach in symptomatic patients.¹⁴

In this study, we organized a prospective follow-up of patients with a diagnosis of myocardial bridge who were symptomatic despite medical treatment. They were selected for surgical therapeutic approach and then followed for 7-81 months (mean 34±2). Myocardial bridges are almost always localized on the LAD. Other coronary arteries are involved rarely. Atherosclerosis in association with myocardial bridge is reported in a few cases. There are some studies maintaining that the proximal part of the bridging segment is prone to atherosclerosis.^{15,16}

In our survey, LAD was the bridged artery in all the cases, and significant atherosclerotic lesions were found in 7(27%) cases: two patients in LAD and five others in LCX and RCA. All the patients underwent surgical unroofing due to unsatisfactory response to medical treatment with beta blockers and calcium channel blockers, and also history of acute coronary syndrome in 46% of cases.

Although it is possible to perform operation through beating heart technique, our recommendation is complete arrest using cardiopulmonary bypass. Accidental opening of the right ventricle can occur during unroofing of the bridged segment of coronary artery, which takes a deep subendocardial course. Reported rate of RV opening in one study is 2 out of 9 cases of supra arterial myotomy.^{17, 18} In our cases, we had this complication in 4 patients.

Myotomy was not successful in one of our patients, and because of recurrent chest pain and significant residual narrowing on the vessel, CABG was carried out and LIMA was grafted to the distal LAD. On follow-up of our patients (7-81 months), only 2 patients developed anginal symptoms without significant residual compression on postoperative angiography; they responded well to medical therapy and there was no major cardiac event, hospital readmission or death and all the

patients survived through out the follow-up period.

We would, therefore, recommend supra-arterial myotomy in that it was successful in reversing myocardial ischemia and increasing coronary flow in 96% of cases and abolishing symptoms in 88% of patients at mid-term follow-up.

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