Myocardial bridge

Surgical outcome and midterm follow up

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ABSTRACT

Objectives: To investigate the results of surgery and long-term follow up in 26 patients who were symptomatic due to myocardial bridge.

Methods: From 1999-2004 more than 18800 coronary angiography were performed in the Shahid Madani Heart Hospital, Tabriz, Iran. Of these, 290 (1.5%) cases had angiographic diagnosis of myocardial bridge. Out of the 290 cases, 26 (9%) patients underwent surgical myotomy for treatment of myocardial bridge causing significant systolic arterial compression. Patients were examined with radio nucleotide study preceding angiography that was positive for ischemia and we found 20 cases (76%). Coronary angiography and left heart catheterization in all patients revealed impaired blood flow due to myocardial bridge in left anterior descending artery and there was an additional atherosclerotic stenosis of coronary arteries in 6 and mitral valve disease in one patient. Supra arterial myotomy was performed in all patients.

Results: We observed 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 left anterior descending despite myotomy and underwent coronary artery bypass graft of left internal mammary artery (LIMA) to distal left anterior descending. During 7-81 months of follow-up (mean 34.2 ± 21), only 2 patients had symptoms of angina that was not shown significant residual compression and symptoms were controlled by medical treatment.

Conclusion: The surgical relief of myocardial ischemia due to myocardial bridge can be accomplished with low operative risk and excellent mid term result.

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Muscle overlying the intramyocardial segment of an epicardial coronary artery is termed myocardial bridge.[1] It is characterized by systolic compression of the tunneled segment which remains clinically silent in vast majority of cases. The angiographic prevalence of bridging has been reported as 0.5 -1.6%.[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 treatment of myocardial bridge. In this study, we report results of surgery and midterm outcome in a series of 26 patients with symptomatic myocardial bridging who underwent supra-arterial myotomy.

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Methods. Between 1997-2004, approximately 18800 coronary angiography were performed in the Shahid Madani Heart Hospital, Tabriz, Iran. We detected 290 cases (1.5%) with angiographic diagnosis of myocardial bridge. Among them, 26 (9%) patients 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%), one patient had recurrent attacks of severe chest pain that need admission to the Coronary Care Unit and one patient had acute anterior myocardial infarction. Characteristics and coronary risk factors are shown in **Table 1**. Myocardial bridges were localized in the middle portion of the left anterior descending (LAD) and were diagnosed by systolic compression and angiographic milking. The diameter of tunneled segment was measured by digital caliper in systole and diastole. The systolic reduction of intraluminal diameter was between 60-80% (average 70 ± 8%) (**Figure 1**). Resting electrocardiogram (ECG) showed ischemic ST-T changes in 11 cases and left ventricular hypertrophy (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 Bruce protocol was performed in all of the patients and was positive in 24 patients (92%). Myocardial perfusion scan with Thallium 201 was also performed in all of the patients that showed reversible myocardial perfusion abnormality in anterior wall and septum; we found 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.

Table1 - Patient's characteristics.

Parameters	Variables	
	n	(%)
Age (years) (mean)	26	48±5
Male	19	(73)
Chest pain	21	(80)
Acute coronary syndrome	12	(46)
Left ventricular hypertrophy	8	(30)
Electrocardiography	20	(76)
Exercise tolerance test (positive)	24	(92)
Myocardial perfusion imaging (positive)	20	(76)
Hypertension	9	(33)
Smoking	8	(30)

Results. Unroofing of the myocardial bridge was performed through median sternotomy. The surgical method in the first 3 patients was without cardiopulmonary bypass (off-pump, beating heart); however, this technique changed to cardiopulmonary bypass because of LAD traumatization in the third patient. In 5 patients, saphenus vein grafts used to bypass diseased segments of atherosclerotic arteries other than LAD. In 2 patients, LIMA was grafted to LAD due to significant atherosclerotic lesion in the first patient, and spontaneous coronary artery dissection in the second one. 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 mortality perioperative and through follow up period. One patient had recurrent anginal chest pain after operation and coronary angiography revealed residual narrowing of LAD despite myotomy that underwent CABG by grafting LIMA to distal of the LAD. Perioperative myocardial infarction was not observed by serial measurement of CKMB and serial ECG. All patients followed between 7-81 months (average 34±2 months). No mortality was observed during the study period. Two cases had recurrence chest pain and the rest had symptom free and all cases were free of major cardiac events. Two symptomatic cases had no apparent systolic compression and symptoms were alleviated by prescribing beta blockers. Exercise test and myocardial perfusion scan were performed in 6-12 months following operation and show no any residual ischemia. Twelve patients underwent postoperative coronary angiography for evaluation of residual narrowing that showed complete resolution of systolic squeezing of the vessel (**Figure 2**).

Discussion. Myocardial bridges are relatively a common finding. Its prevalence differs on the basis of the study method, with a much higher rate at autopsy (50%)[4] versus angiography (0.5-1.6%). Although in many cases, only asymptomatic finding was presence.[5,6] They were diagnosed in vivo by angiography. Through new imaging techniques such as intravascular ultrasound (IVUS) and intracoronary Doppler ultrasound (ICD) it has been known more about the mechanism of ischemia and morphological and functional features of myocardial bridging.[7] In literature, there are different treatment options to improve quality of life in symptomatic patients although hard evidence for a favorable effect on

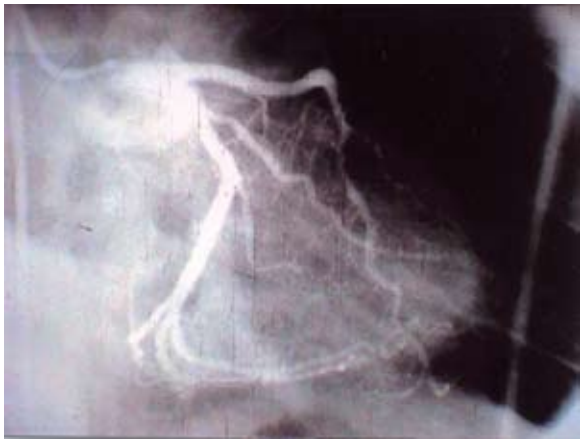

Figure 1 - Coronary angiography shows myocardial bridge of the left anterior descending artery with 80% systolic compression before surgery.

Figure 2 - Coronary angiography shows no systolic compression after supra arterial myotomy.

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 et al[9] suggested that surgical unroofing of the coronary artery can prevent sudden death. Sorajja et al[10] observed no increased risk of death, including sudden cardiac death, among adult patients with HCM who had myocardial bridge. In subjects refractory to medication, surgical myotomy, first reported by Binet et al,[11] abolishes clinical symptoms and is associated with reversal of local myocardial ischemia and an increase in coronary flow.[12] In 1995, Stables et al[13] first reported coronary stenting as an interventional approach to severe myocardial bridging refractory

Table 2 - Type of surgery and results and complications.

Type of surgery		Results and complications	
Supra arterial myotomy	26	Symptom free	23
CABG	7	Recurrent chest pain	3
		Inadvertent RV opening	4
OMVC	1	LAD traumatization	1

CABG - coronary artery bypass graft, OMVC - open mitral valve commissurotomy, LAD - left anterior descending

to medication with successful short term results. However, approximately 50% of these patients developed restenosis and major periprocedural complications that coronary intervention is not a generally recommended approach in symptomatic patients.[14] In this study, we organized a prospective follow up of patients with diagnosis of myocardial bridge who underwent surgical unroofing due to unsatisfactory response to medical treatment with beta blockers and calcium channel blocker, and also the history of acute coronary syndrome in 46% of cases. Myocardial bridges are almost always localized on LAD. Other coronary arteries are involved rarely. Atherosclerosis in association with myocardial bridge is reported in a few cases. There are some studies express that 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 lesion found in 7 (27%) of cases; 2 patients in LAD and 5 others in LCX and RCA (**Table 2**). 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 could happen during unroofing of bridged segment of the coronary artery that takes a deep subendocardial course. Reported rate of the right ventricle 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 (**Table 2**). Myotomy was not successful in one of our patients because of the recurrent chest pain and significant residual narrowing on the vessel. We carried out CABG and grafted LIMA to distal LAD. On follow up period of our patients (7- 81 months) only 2 patients developed anginal symptoms without significant residual compression showed on postoperative angiography and responded well to the 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 concluded that supra arterial

myotomy was successful in reversal of myocardial ischemia and increase in coronary flow in 96% of cases and abolished symptoms in 88% of patients at midterm follow up and can be recommended.

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References

1. Nayar PG, Nyamu P, Venkitachalam L, Ajit SM. Myocardial infarction due to myocardial bridging. ***Indian Heart J*** 2002; 54: 711-712.
2. Nobel J, Bourassa MG, Petitclerc R, Dyrda I. Myocardial bridging and milking effect of the left anterior descending coronary artery: normal variant or obstruction? ***Am J Cardiol*** 1976; 37: 993-999.
3. Achrafi H. Hypertrophic cardiomyopathy and myocardial bridging. ***Int J Cardiol*** 1992; 37: 111-112.
4. Polaek P, Zechmeister A. The occurrence and significance of myocardial bridging causing coronary artery obstruction. ***Scand J Thorac Cardiovasc Surg*** 1992; 26: 107-111.
5. Rossi L, Dander B, Nidasio GP, Arbustinin E, Paris B, Vassanelli C, et al. Myocardial bridges and ischemic heart disease. ***Eur Heart J*** 1980; 1: 239-245.
6. Arnau Vives MA, Martinez Dolz LV, Almenar Bonet L, Lalaguna LA, Ten Morro F, Palencia Perez M. Myocardial bridging as a cause of acute ischemia. Description of a case and review of literature. ***Rev Esp Cardiol*** 1999; 25: 441.
7. Ge J, Jeremias A, Rupp A, Abels M, Baumgart D, Liu F, et al. New signs characteristic of myocardial bridging demonstrated by intracoronary ultrasound and Doppler. ***Eur Heart J*** 1999; 20: 1707-1716.
8. Mohlenkamp S, Hort W, Ge J, Erbel R. Update on myocardial bridging. ***Circulation*** 2002; 106: 2616-2622.
9. Yetman AT, McCrindle BW, MacDonald C, Freedom RM, Gow R. Myocardial bridging in children with hypertrophic cardiomyopathy: a risk factor for sudden death. ***N Engl J Med*** 1998; 339: 1201-1209.
10. Sorajja P, Ommen SR, Nishimura RA, Gersh BJ, Tajik AJ, Holmes DR. Myocardial bridging in adult patients with hypertrophic cardiomyopathy. ***J Am Coll Cardiol*** 2003; 42: 889-894.
11. Binet JP, Planche C, Leriche H, Raza A, Kone A, Piot C, et al. Myocardial bridge compressing the anterior inter-ventricular artery. Apropos of a successfully operated case. ***Arch Mal Coeur Vaiss*** 1975; 68: 85-90. French.
12. Hill RC, Chitwood WR Jr, Bashore TM, Sink JD, Cox JL, Wechsler AS. Coronary flow and regional function before and after supraarterial myotomy for myocardial bridging. ***Ann Thorac Surg*** 1981; 31: 176-181.
13. Stables RH, Knight CJ, McNeill JG, Sigwart U. Coronary stenting in the management of myocardial ischemia caused by muscle bridging. ***Br Heart J*** 1995; 74: 90-92.
14. Haager PK, Schwarz ER, Vom Dahl J, Klues HG, Reffelmann T, Hanrath P. Long-term angiographic and clinical follow up in patients with stent implantation for symptomatic myocardial bridging. ***Heart*** 2000; 84: 403-408.
15. Masuda T, Ishikawa Y, Akasaka Y, Itoh K, Kiguchi H, Ishii T. The effect of myocardial bridging of the coronary artery on vasoactive agents and atherosclerosis localization. ***J Pathol*** 2001; 193: 408-414.
16. Ge J, Erbel R, Gorge G, Haude M, Meyer J. High wall shear stress proximal to myocardial bridging and atherosclerosis: intracoronary ultrasound and pressure measurements. ***Br Heart J*** 1995; 73: 462-465.
17. Iversen S, Hake U, Meyer E, Erbel R, Diefenbach C, Oelert H. Surgical treatment of myocardial bridging causing coronary artery obstruction. ***Scand J Thorac Cardiovasc Surg*** 1992; 26: 107-111.
18. Ochsner JL, Mills NL. Surgical management of diseased intracavitary coronary arteries. ***Ann Thorac Surg*** 1984; 38: 356-362.