Original Article

Clinical and Angiographic Characteristics of Myocardial Bridges: a Descriptive Report of 19 Cases and Follow-up Outcomes

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Received 16 December 2006; Accepted 13 March 2007

Abstract

Background: *Muscle fibers overlying the intramyocardial segment of an epicardial coronary artery are termed myocardial bridge (MB). The aim of this study was to analyze the mid-term outcome of MB and to examine its possible association with angiographic findings and concomitant cardiac pathologies such as hypertrophic cardiomyopathy (HCM).*

Methods: *From a total of 3218 patients admitted for coronary angiography during 9 consecutive months, 28 (0.9%) were diagnosed with MBs with stenoses≥50%. Of these, 19 referred for follow-up with a median duration of 18 months.*

Results: *HCM was present in 5 patients (26.3%), of whom 4 had MB as the sole finding in angiography. Of the 19 patients, 14 had diastolic dysfunction. In follow-up, 2 patients were treated with revascularization strategies due to the concomitant coronary artery disease and in 2, syncope occurred. For two patients, an intra-cardiac device and a permanent pacemaker were implanted. Three patients with MB as the sole finding in angiography were readmitted because of chest pain.*

Conclusion: *Diastolic dysfunction may contribute to the presentation of symptoms of muscle bridging. Also, myocardial bridging as the only finding in coronary angiography is highly associated with hypertrophic cardiomyopathy and may help to detect this group of patients. The mid-term outcome of myocardial bridges is favorable.*

J Teh Univ Heart Ctr 2 (2007) 105-110

Keywords: *Myocardial infarction • Coronary artery disease • Hypertrophic cardiomyopathy*

Introduction

Muscle overlying the intramyocardial segment of an epicardial coronary artery, first mentioned in 1737[1] and described angiographically in 1960[2] is termed a myocardial bridge (MB). This situation is characterized by the decrease in the coronary blood flow during systole due to the compression of the myocardial fibrils surrounding the epicardial coronary artery in a certain segment. Autopsy studies have found a frequency of 15 to 85 percent,[3-5] while angiographic studies have noted a lower incidence of myocardial bridging ranging from 0.5 to 33 percent.[6-8] It has been suggested that bridging rarely causes myocardial ischemia and this could partly explain such discrepancies.[9] On the other hand, the wide variability in the incidence of different anatomicopathological series seems to depend on the skill of the operators performing the

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dissection procedures.[10]

Several diseases have been reported concomitantly with myocardial bridges. For instance, previous literature has shown the incidence of myocardial bridging to be 28 to 40 percent in children with hypertrophic cardiomyopathy (HCM).[11,12] The presence of MB has also been known as an important cause of myocardial ischemia in HCM.[13,14]

This study was designed to analyze the mid-term outcomes of patients with myocardial bridges in a series of 19 cases and to describe the possible association between myocardial bridge, demographic and angiographic findings, and concomitant diseases like hypertrophic cardiomyopathy.

Methods

The study population was selected from among a total of 3218 patients with suspected coronary artery disease (CAD) admitted to Tehran Heart Center for coronary angiography between July 2004 and February 2005. Coronary compression was defined as a maximum systolic compression ≥50%. In this population, 28 patients had systolic compression ≥50%, from whom only 19 referred for follow-up. The remaining 9 patients were missed because of geographic diversity and/or residential change, and inaccessibility of their new addresses. Hence, this study population included 19 patients. All angiograms were assessed by two independent reviewers unaware of the patients' clinical status. In cases of controversy, the opinion of a third cardiologist was sought. Left ventricular ejection fraction (LVEF) was measured by the quantitative two dimensional (biplane Simpson) method. Patients were defined as having systolic dysfunction if their LVEF was 50%. Patients with an LVEF>50% were further examined for diastolic dysfunction. Each of these patients underwent pulsed wave Doppler examination of mitral inflow before and during the Valsalva maneuver. Diastolic dysfunction was categorized according to the progression of diastolic dysfunction as normal; mild (grade 1), defined as impaired relaxation without evidence of increased filling pressures; moderate (grade 2), defined as impaired relaxation associated with moderately increased filling pressures or pseudonormal filling; and severe (grades 3 and 4), defined as reversible restrictive or fixed restrictive patterns.[15] The HCM cases were confirmed by two cardiologists according to the clinical, electrocardiographic, and echocardiographic evidence, and reconfirmed by a third cardiologist in controversial cases. A cardiologist performed repeated echocardiography and completed the follow-up questionnaires in the 18-month follow-up duration. Results of exercise thallium scintigraphy were evaluated when available, and images were analyzed qualitatively in the anterior, apical, inferior, septal, and lateral regions. Holter recordings were analyzed for the detection of ventricular tachycardia. Problems in follow-up included readmission, coronary artery bypass grafting (CABG), percutaneous coronary intervention (PCI), syncope, and death. Informed consent was obtained from all the patients before enrollment into this study. The procedures followed were in accordance with ethical standards of the Tehran Heart Center Ethics Committee.

Results

Demographic findings

Selective demographic and clinical characteristics are shown in Tables 1 and 2. Of the 19 patients with angiograms judged adequate for the diagnosis of myocardial bridging, 18 were male and 1 was female. The mean age±standard deviation was 52.6±12.7 years (range: 28-72 years). As shown in Table 1, two patients had diabetes mellitus, 7 had systemic hypertension, 9 had hyperlipidemia, 3 had family history of sudden cardiac death, and 5 were active smokers at the time when coronary angiography was performed. Symptoms at presentation included stable angina in 7, unstable angina in 6, 2 anterior and 1 inferior myocardial infarction (MI) in 3, palpitation in 1, and syncope in 2. The indications for coronary angiography were: positive exercise tolerance test in 6, MI in 2, positive perfusion scans in 3, syncope in 2, and high clinical suspicion for CAD in the remaining 6. In 5 of these cases (26.3%), HCM was present. From among 11 patients who had myocardial bridging as the sole finding in coronary angiography (without HCM or significant CAD), 5 had presented with stable angina, 5 with unstable angina, and 1 with anterior MI. In this population, no one had presented with syncope or palpitation.

Echocardiographic, scintigraphic, and angiographic findings

All the myocardial bridges were detected in the mid portion of the LAD. Table 2 shows that out of the 19 patients, 8 had left ventricular hypertrophy and 14 had diastolic dysfunction. Myocardial perfusion scan was performed for 3 patients, which showed inferior ischemia in all of them. No reverse redistribution was detected. Two of these patients had presented with chest pain and one with syncope. In all of these patients, myocardial bridge was the only angiographic finding. All of these patients had diastolic dysfunction, and the length of MB fragment in them was 20-25 mm. The lengths of myocardial bridges in the total population were less than 10 mm in one, 10-20 mm in 13, and above 20 mm in the remaining 5 patients. The severity of systolic compression was about 50% in 9, over 50% to 70% in 6, 70%-90% in 3 patients, and over 90% in 1 patient. One of the patients had left main track involvement. In 3 patients, the left circumflex artery was dominant. In one case, there was co-dominancy; and in the remaining cases, the right coronary artery was

dominant. In all of the cases with myocardial bridge, the mid-portion of LAD was affected.

Table 1. Demographic characteristics (n=19)

Age (yr)	*52.6±12.7
Male	18 (94.7%)
Hypertension (mm Hg)	9 (47.4%)
Diabetes mellitus	2 (10.5%)
Hyperlipidemia	7 (36.8%)
Cigarette smoking	5 (26.3%)
Family history of coronary artery disease	8 (42.1%)
Family history of sudden cardiac death	3 (15.8%)

*Mean±standard deviation

Table 2. Clinical and angiographic characteristics (n=19)

Left ventricular hypertrophy	8 (42.1%)
Diastolic dysfunction	14 (73.7%)
Dominancy	
Right coronary artery	15 (78.9%)
Left circumflex	3 (15.8%)
Vessel involvement	
Myocardial bridge	14 (73.7%)
Myocardial bridge + 1-vessel disease	1 (5.3%)
Myocardial bridge + 2-vessel disease	3 (15.8%)
Ejection Fraction (%)	60.2±5.9*
Severity of MB stenosis (%)	60±14.5*
Length of MB (mm)	19.6±4.9*

*Mean±standard deviation
MB, Myocardial bridging

Follow-up data

The median follow-up duration was 18 months (range: 12-23 months). In the follow-up period, one patient underwent CABG, another one was treated with PCI based on angiographic results, and in 2 patients syncope occurred. The causes for syncope were transient complete heart block and ventricular tachycardia with underlying hypertrophic cardiomyopathy. Four patients were readmitted because of chest pain. In two patients, an implantable cardioverter defibrillator (ICD) and permanent pacemaker (PPM) were implanted. All the patients received beta-blockers, and some patients took diltiazem as well. All of the 11 cases without HCM or CAD were followed by medical therapy.

Discussion

Our study is a descriptive report of baseline and angiographic characteristics in a series of 19 patients who had been diagnosed with myocardial bridging. The angiographic appearance of myocardial bridges depends on several factors, including the length of myocardial bridge.[16] The use of provocation tests may enhance systolic myocardial compression and thereby reveal myocardial bridges in 40% of cases.[17] In this study, the lengths of myocardial bridges were between 10 and 25 mm, which were within the typical range.16 Moreover, like the Lozano's study,[18] intracoronary nitroglycerine was not administered systematically, except when a possible reduction in the vessel diameter was detected at some point in the coronary tree. On the other hand, we only included myocardial bridges with at least 50% stenosis. These factors may account for the relatively low prevalence of myocardial bridging in our study population (0.9%) compared to other studies.

In our study, the segment that was affected was only the mid-portion of the LAD, although some cases have been found in other coronary segments,[19,20] It has been reported that the compressed segment is frequently spared from atherosclerotic changes.[2,3] Interestingly, we found atherosclerotic plaques neither in the territory of the bridge nor in the distal LAD.

The latter finding is consistent with a previous report.[21] One of the striking results was that diastolic dysfunction presented in as much as 73.7% of our study population. Normally, only 15% of the coronary blood flow occurs during systole, and because myocardial bridging is a systolic event on angiography, its clinical significance and relevance have been questioned.[22] It has been proposed that although arterial compression takes place during systole, there is also inadequate coronary flow in the first third of diastole, probably leading to a decreased reserve of coronary flow and lower-than-normal ischemia threshold.[23] However, this flow reserve is sufficient in baseline situations and ischemia is only manifested in situations like increased oxygen demand.[24,25] Our finding may be of particular importance, suggesting that in addition to these high-oxygen demanding situations, diastolic dysfunction may also play a role in making the patients symptomatic. Diastolic dysfunction may predispose patients to symptomatic myocardial bridging through the following mechanisms: First, if relaxation is excessively slow, it can elevate not only early but also late diastolic pressures, particularly at faster heart rates such as during exercise;[26] as left ventricular pressure increases in diastolic dysfunction, the myocardial perfusion pressure drops. Second, because of the delay in myocardial relaxation, a part of the compression produced in the early diastole persists through the whole diastole[25] and causes further reduction in the blood flow to the myocardium; Furthermore, after a vessel is compressed during systole, it is refilled with blood during diastole, and the intra-luminal volume of the vessel is increased. This refilling causes a delay in the blood flow during diastole; hence myocardial perfusion further decreases. The longer the duration of decompression during diastole is, the clearer the effect of this delay will be. This may be a reason why myocardial bridging, albeit a congenital condition, becomes symptomatic in adults. This is contrary to the fact that in

lower ages, the heart beats faster than the adults, and the time that the heart remains in systole is longer. According to some authors, the presence of tachycardia could unmask the ischemic effect of a myocardial bridge by decreasing the diastolic filling time and coronary flow reserve and increasing the importance of systolic blood flow.[27] For all these reasons, it is hypothesized that some additional factors such as diastolic dysfunction may contribute to the adverse outcomes of systolic compression.

The relationship between the degree of systolic compression and the clinical symptoms has been debated in many previous studies, either showing the presence of an association28 or no association.[9] In our study, the degree of systolic compression was between 50 and 90 percent. We did not find any correlation between the degree of vessel compression and the presenting symptoms. However, this may be partly due to the small sample size.

In our study, out of the 11 patients who had only myocardial bridging in coronary angiography, 5 had stable angina, 5 had unstable angina, and 1 presented with MI. Other studies have reported stable or unstable angina as frequent, and myocardial infarction, ventricular tachyarrhythmia, and cardiac death as infrequent clinical presentations.[29-31] Be that as it may, considering the prevalence of myocardial bridging, these complications are rare.

Therapy in symptomatic patients, may improve quality of life, although hard evidence for a favorable effect on morbidity and mortality is missing. Three treatment strategies have been explored: (1) negative inotropic and/or negative chronotropic agents, like beta-blockers and calcium antagonists; (2) surgical myotomy and/or CABG and (3) stenting of the tunneled segment.19 In our study, all the patients were medically treated with beta-blockers, and in some cases, with diltiazem. Only one of our patients, who had concomitant coronary artery disease, underwent surgical un-roofing. This patient had severe systolic compression with signs refractory to medical treatment, which is an indication for surgical intervention, according to some previous Reports.[32,33] Some authors have shown improved prognosis with the surgical division of the bridge[34] with low operative risks and excellent mid-term results.[35]

Conflicting observations have been reported on the prognosis of myocardial bridges. Some reports have shown an excellent long-term prognosis with angiographically detected, isolated bridges.[36] Overall, long-term prognosis in patients with isolated myocardial bridging is generally good. In two studies on the long-term outcomes, no myocardial bridge-related MI or death was reported in patients with otherwise normal coronary arteries.[27,37] In the latter study, among 21 patients monitored for 3.4 years, two patients with coexistent CAD experienced MI infarction and underwent CABG. All the other patients, including 7 with HCM and 8 with normal coronaries remained event-free. In our study, CABG and PCI were performed for 2 patients, as recommended by coronary angiography and the established coronary artery disease. Two patients experienced syncope at follow-up, and 4 of them were readmitted, of whom, one had HCM. Two other patients with HCM developed syncope at follow-up, for whom ICD and PPM were implanted. We observed that three patients with myocardial bridging as the only finding were readmitted to the hospital. This could show that the events at follow-up could be more correlated with the severity of concomitant vessel involvement and/or HCM rather than the presence of myocardial bridge, suggesting that myocardial bridges may have excellent mid-term outcomes.

Myocardial bridging is most often considered to be a congenital anomaly, and concomitant cardiac pathologies, such as hypertrophic cardiomyopathy, have also been reported.[11-14] In our study population, 5 patients (26.4%) had HCM with an age range of 44-55 years old. In 4 of them, we found myocardial bridging as the sole underlying coronary finding. Indeed, myocardial ischemia is an important aspect of the pathophysiology for HCM patients, but significant coronary artery stenosis is rarely noted. This may be an important finding, as it has been shown that adverse cardiac events such as sudden death, ventricular tachycardia, and impaired exercise test may occur as a result of myocardial bridging in cases with HCM ( ). HCM in many instances is present as a genetic mutation, and may remain clinically undiagnosed. A study on the genetic mutations in myocardial bridging may lead to the identification of genotypes of HCM.

The results of this study need to be confirmed by further studies with larger study populations. Moreover, as this study was performed retrospectively, it was not possible to match the patients in the angiographic performance and medical options. This problem can influence the identification and selection of patients and also the results of follow-up. Finally, we did not use intravenous nitroglycerine. This may have caused us to miss possible, but infrequent, cases of myocardial bridges in the distal parts.

Conclusion

In our setting, the frequency of myocardial bridging, although low, was within the general range. We detected a high percentage of cases of diastolic dysfunction in these patients, and hypothesized that this factor may contribute to the presentation of symptoms in this condition. Also, the possibility of a high correlation between myocardial bridging and presentation of symptoms in hypertrophic cardiomyopathy is suggested.

Acknowledgement

This study was supported by Tehran Heart Center, Tehran University of Medical Sciences. The authors declare that

they do not have any conflicts of interests.

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