Long-Term Prognosis of Non-Interventionally Followed Patients with Isolated Myocardial Bridge and Severe Systolic Compression of the Left Anterior Descending Coronary Artery

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ABSTRACT

Purpose: The aim of this study was to investigate the long-term prognosis of non-interventionally followed patients with myocardial bridge and angiographic milking of the left anterior descending (LAD) coronary artery.

Methods: All of the coronary angiography records from May 2000 to November 2007 were reevaluated and patients who had more than 70% narrowing during systole on LAD were eligible for the present study. Follow-up was carried out by physical examination, echocardiography, and treadmill exercise testing. The clinical situations of the patients, medical treatment at the time of follow-up, and experienced events (death, myocardial infarction, or revascularization) were recorded.

Results: There were 59 eligible patients (44 male, 74.6%). The mean age of the patients was 54 ± 11 years. The bridges were located in the proximal, mid, and distal portion of the LAD in 17 (28.8%), 20 (33.9%), and 22 (37.3%) patients, respectively. Distributions of the narrowing degree were as follows: between 70% to 89% in 33 (56%) patients and 90% to 100% in 26 (44%) patients. Mean follow-up duration of the group was 37 ± 13 months (range 15–65 mo). The clinical presentation during follow-up was stable angina in 9 (15.3%) cases, atypical angina in 12 (20.3%), atypical chest pain in 13 (22%), dyspnea in 3 (5.1%), and syncope in 3 (5.1%) cases. There were no experienced events and/or hospitalizations related to cardiac disease. Echocardiographic examination revealed normal systolic ventricular function. Only 17 (28.8%) patients continued to use medication. Most of them were on β-blocker therapy.

Conclusion: Patients with myocardial bridges and angiographic milking of the LAD coronary artery have a good long-term prognosis.

Introduction

Myocardial bridging is anatomically defined as the muscle overlying the intramural segment of a major epicardial coronary artery. A myocardial bridge is the most common congenital coronary anomaly, with an incidence of between 1.5% and 16% as assessed on coronary angiography and up to 80% as assessed at necroscopy.[1–3] It is usually considered an innocent anomaly.[4–6] Although it is rare, myocardial infarction, left ventricular dysfunction, paroxysmal atrioventricular block, as well as exercise-induced ventricular tachycardia and sudden cardiac death are accused sequela of this pathology.[4–12] The aim of this study was to investigate the prognosis of non-interventionally followed patients with myocardial bridge and angiographic milking of the left anterior descending (LAD) coronary artery.

Material and Methods

The cardiac catheterization laboratory data registry was used to identify patients with a myocardial bridge in the LAD coronary artery. All of the coronary angiography records from May 2000 to November 2007 were reviewed and the patients who had more than 70% narrowing in the LAD during systole have been accepted into the study.

Exclusion Criteria

The patients who had the following criteria were not included in the study: (1) existence of accompanying coronary artery disease that depict as fixed stenosis of more than 50% at some point of the coronary tree at coronary angiography; (2) patients who have hypertrophic cardiomyopathy or valvular heart disease in 2-dimensional echocardiography; (3) patients with congestive heart failure and/or systolic dysfunction in echocardiographic assessment not related to myocardial bridge; and (4) myocardial bridges located in the circumflex or right coronary artery and cause less than 70% narrowing during systole were excluded.

We obtained coronary angiography in routine fashion using Judkins technique in multiple projections, including cranial and caudal angulations with left anterior oblique

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and right anterior oblique projections. Our radiographic equipments are the Siemens Axiom Artis and Philips H 3000 systems (Siemens, Berlin, Germany). Cine frames were selected from the projections in which the phenomenon of systolic compression of the LAD coronary artery could be observed clearly. Intracoronary nitroglycerin administration was not given in most of the patients. The 2 trained observers evaluated the lesions together. Quantitative analysis was done in order to detect the degree of systolic narrowing of the coronary artery.

Follow-Up

Follow-up was carried out by physical examination, echocardiography, treadmill exercise test, and/or myocardial perfusion scintigraphy. The clinical status of the patients, medical treatment at the follow-up, and events experienced (death, myocardial infarction, or revascularization) were recorded.

Results

A total of 9284 patients underwent coronary angiography during the study period, among them 28.8% (2671 patients) were found to have normal coronary arteries. After exclusion of patients with associated cardiac pathologies like hypertrophic cardiomyopathy or valvular heart disease, 59 patients (0.64%) showed a systolic compression of more than 70% in the LAD coronary artery with otherwise normal coronary arteries. All of them were followed with echocardiography and exercise stress testing. None of the patients had a coronary intervention like stenting or cardiovascular surgery.

The mean age of the patients was 54±11 years. Clinical characteristics of the study group were shown in Table 1. The bridges were located in proximal, mid, and distal portions of the LAD in 17 (28.8%), 20 (33.9%), and 22 (37.3%) patients, respectively. Mean narrowing during systole was 85%±9%. Distributions of the narrowing degree were as follows: between 70% to 89% in 33 (56%) patients and 90% to 100% in 26 (44%) patients. Symptomatic patients had more severe lesions and showed a tendency to have a more proximally located bridge. A total of 6 of the 17 (35.2%) patients with proximal bridge had a symptom, whereas 9 of the 42 (21.4%) with a nonproximally located bridge had a symptom. The mean stenosis during systole was 89% ± 9% in symptomatic patients vs 84%±9% in asymptomatic patients ($P = 0.049$).

Follow-Up

Mean follow-up duration was 37±13 months (range 15–65 mo). The clinical presentation during follow-up was shown in Table 2. There was no death, new acute coronary syndromes, clinical congestive heart failure, and/or hospitalization related to cardiac disease. Echocardiographic examination revealed normal systolic ventricular function. The mean ejection fraction was 70%±7%. There was only 1 case with moderate ventricular dysfunction who had an acute anterior myocardial infarction at the time of index presentation. All other patients' ventricular function, left ventricular diastolic, and systolic dimensions were in normal limits (49±4 mm and 28±5 mm, respectively). All patients underwent a treadmill exercise test according to Bruce protocol (Table 2). Four patients experienced chest pain during the exercise, however ischemic ST-segment depression was observed in only 1 of them. One patient showed ST-segment

Table 1. Clinical Characteristics of the Patients at the Time of Coronary Angiography

Clinical Characteristics	n (%)
Male	44 (74.6%)
Diabetes mellitus	12 (20.3%)
Hypertension	29 (49.2%)
Hyperlipidemia	20 (33.8%)
Family history (CAD and/or SCD)	8 (13.4%)
Active smoker	31 (52.5%)

Abbreviations: CAD, coronary artery disease; SCD, sudden cardiac death.

Table 2. Clinical Status of the Patients During the Follow-Up Period

	n (%)
Clinical Status	
Stable angina pectoris	9 (15.3%)
Atypical angina pectoris	12 (20.3%)
Atypical chest pain	13 (22%)
Dyspnea	3 (5.1%)
Syncope	3 (5.1%)
Asymptomatic	19 (32.2%)
Treadmill Exercise Test	
Duration (sec)	11.4 ± 2.1
Maximal heart rate (bpm)	156 ± 13
Maximal blood pressure (mm Hg)	174 ± 11
Duke score	8.7 ± 2.6
Chest pain	4 (6.8%)
ST-segment depression	2 (3.4%)
Arrhythmia	1 (1.7%)

depression without chest pain and 1 patient developed a supraventricular tachycardia.

Only 17 (28.8%) patients continued to use antianginal medication and most of them (15 patients) were on β-blocker therapy. However there were only 11 (18.6%) patients having typical angina and/or ischemia on the treadmill exercise test. Of these, 5 patients (45.4%) were not on any medical treatment.

Discussion

The first definition of myocardial bridge in the literature was in 1960.[13] The prevalence of this abnormality was reported between 0.5% to 12% in angiographic studies.[14,15] Most of the cases are asymptomatic, which explains why the frequency of this anomaly in postmortem study samples is much higher than in angiographic diagnoses. Angiographic appearance is related to the length and thickness of the overlying myocardial segment. In this study, incidence of myocardial bridge in LAD with milking phenomenon prevalence was 0.64% after exclusion of associated cardiac pathologies. In a comparable angiographic cohort, a similar prevalence of 0.42% has been reported.[16]

Although it is generally a benign pathology, sometimes it may result in symptoms like angina, arrhythmias, heart failure, and sudden death. Angina, dyspnea, and syncope were detected in 15.3%, 5.1%, and 5.1% of our patients, respectively. Several mechanisms like systolic obstruction, delayed diastolic relaxation, and vasospasm have been proposed as a cause of clinical symptoms. However it is not clear whether a relationship exists between the degree of reduction in vessel diameter during systole and clinical symptoms.[17] Besides, although arterial compression occurs during systole in patients with myocardial bridge, intravascular ultrasound (IVUS) and Doppler studies revealed that they do not have an adequate blood flow in the first third of the diastole.[18,19] In our study, we found a relationship between symptoms and lesion severity during systole and a probable relation to the location of the stenosis.

We had only 1 patient who had a myocardial infarction at the time of index presentation. No other patient developed a myocardial infarction during the mean 37 months follow-up in these selected myocardial bridge population. On occasion, segmental hypokinesia could be observed due to an ischemia without infarction. However, we did not observe any wall motion abnormality except for the infracted patient. Minimum ejection fraction was 55% among the rest of the patients.

Medical, percutaneous transluminal coronary angioplasty (PTCA), and surgical treatment options are available in patients with proven ischemia. β-Blockers are the suggested medical treatment option as they prolong the diastolic time and decrease the contraction force above the coronary artery.[16] The role of the calcium antagonists is not clear and they are not drugs of first choice, but cases refractory to the β-blockers that responded to diltiazem have been reported.[16] The duration of the need for medical treatment is a point that has not been studied in any series. In our group, nearly a one-third of the patients continued to use medical therapy and a great majority of them were on β-blocker therapy.

Percutaneous coronary intervention has been suggested as an alternative therapeutic option. Nevertheless, the results of balloon angioplasty are unsatisfactory, because elastic recoil limits the control of symptoms. Haager et al.[20] described implantation of intracoronary stents in 11 severely symptomatic patients. Severe stenosis was developed in 5 patients, 4 of them required reintervention. A stent fracture was reported in a case report.[21] Stent implantation should not be considered as the treatment of choice because of high complication and restenosis rate.

Another alternative treatment to relieve compression on the vessel is surgical myectomy.[22] It improves blood flow and ischemia. However a deep incision is required for a deep bridge and the complication rate is higher than any other intervention. It can be performed in severely ischemic patients, but the prognostic value is limited.

Cases of revascularization using the internal mammary artery have also been described.[23] Although the long-term prognosis is good; it should be done only in selected cases.

Medical therapy should be the first and principal strategy, and interventions should be limited to patients with refractory angina despite medical therapy.[24]

In our study, long-term prognosis in patients with isolated myocardial bridge was quite good. We have not observed any major coronary event in nearly 3 years follow-up. Only 1 patient had a myocardial infarction at the time of index presentation. Event-free survival was 98% with a noninterventional approach. Even if only one-third of our patients were on medical treatment, only 18.6% of the patients had typical angina and/or a positive treadmill test. Therefore it seems following-up patients with medical treatment is the only reasonable approach in a selected high risk group of isolated myocardial bridge and severely systolic compression of the LAD coronary artery.

Limitations of the Study

In this study, the patients have been included from the databases of cardiac catheterization laboratories. Therefore we cannot put forward an idea about the prognosis of all myocardial bridge patients. However, we think that this selected group has the highest risk for major cardiac events.

The myocardial bridge is more prominent when intracoronary nitroglycerin is given. In our study, intracoronary nitroglycerin was not administered systematically. However, all our patients had a severe systolic compression without need of provocation.

Conclusion

Patients with myocardial bridge and angiographic systolic milking of the LAD coronary artery have a good long-term prognosis. Medical treatment seems to be sufficient to control the symptoms. Moreover, in some cases, continuous therapy was really unnecessary. Of the two-thirds of the patients who did not continue to take medicine, only one-fifth had a myocardial ischemia.

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