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The Incidence and Significance of Myocardial Bridge in a Prospectively Defined Population of Patients Undergoing Coronary Angiography for Chest Pain

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Since its first description in 1922, the incidence and pathophysiologic significance of myocardial bridges has remained controversial due to the relatively small size and retrospective design of most studies. We assessed the incidence and clinical consequence of myocardial bridges in 2547 patients undergoing coronary arteriography over a 16-month period at our medical center. Of the 511 patients without fixed coronary obstruction, 26 (5%) were found to have myocardial bridge. Of the 26 patients only 1 demonstrated ischemia as assessed by Tc-99m MIBI myocardial perfusion scintigraphy during treadmill exercise testing in the distribution of the culprit lesion. Therefore, the results of the present study suggest that angiographically detectable myocardial bridges are uncommon in patients undergoing routine angiography and are associated with ischemia in only one patient.

Key words : Myocardial bridge, Angiography, Chest pain

INTRODUCTION

The coronary arteries and their major branches travel along the surface of the heart under the epicardium. However, occasionally, a portion of these arteries may be embedded in muscle; a finding that has been variously described as a mural coronary artery, submerged coronary artery, or more frequently, myocardial bridge [5, 7]. First described by pathologists in the early 1920's, myocardial bridges appear almost exclusively in the left anterior descending coronary artery [6]. Angiographically, myocardial bridges were characterized by systolic compression of a portion of the coronary artery. While the incidence of myocardial bridging has been reported between 15 and 85% in pathologic series, angiographic evidence is substantially less (0.51 to 2.5%) [2, 9, 15, 16, 17]. Moreover, the pathophysiologic significance of a myocardial bridge remains controversial. While some reports suggest that myocardial bridges might have "protective effects" on the coronary artery at the site of the bridging, other studies suggest a relationship between the presence of a myocardial bridge and symptomatic ischemia [2, 3, 5, 7, 14, 15]. Indeed, the presence of a myocardial bridge has been associated with a myocardial infarction and possible coronary vasoconstriction [4].

Unfortunately, studies assessing both the incidence and significance of myocardial bridges have been relatively small [1, 2, 4, 5, 6, 7, 8, 11, 12, 16, 17]. Furthermore, in most cases these studies have not assessed the hemodynamic or functional significance of the myocardial bridges. Therefore, the present study was performed to assess both the incidence and relevance of myocardial bridging in a large population of patients admitted to our medical center for evaluation of anginal symptoms.

PATIENTS AND METHODS

We prospectively evaluated 2,547 consecutive patients seen in our medical center between April 1995 and August 1996 admitted with a diagnosis of possible coronary

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artery disease requiring diagnostic coronary arteriography. Patients were defined as having either typical or atypical angina. Patients with typical angina pectoris described substernal pressure or pain of brief duration that occurred during exercise and was relieved by rest. Patients were considered to have atypical angina if their chest pain was longer than 15 min in duration (without evidence of myocardial infarction) and/or occurred mainly at rest.

All patients underwent an echocardiogram and routine pre-procedure care prior to a left heart catheterization with coronary angiography using standard procedures. Patients were excluded from further study if they were found to have hemodynamically significant coronary artery disease as evidenced by coronary angiography, valvular heart disease as defined by echocardiography, or a cardiomyopathy as defined by an ejection fraction by echocardiography of less than 30%. Each cineangiogram was reviewed by at least two qualified cardiologists who evaluated the angiogram and measured coronary lumen diameters were double-blinded. Arteriographic quantification of systolic lumen compression was performed using a programmable digital caliper to measure the systolic lumen diameter reduction and the length as previously described [12]. Measurements were performed in the left anterior oblique position and included systolic lumen diameter reduction and length. Measurements were only accepted when the disparity between the measurements of the two investigators was less than 20%.

Measurements were performed using the following equations:

1) Lumen diameter reduction %
   $100 - [2m(D1 + D2) \times 100]$
   m: minimal lumen diameter in systole.
   D1: diastolic diameter proximal to the obstruction.
   D2: diastolic diameter distal to the obstruction
2) Actual length of systolic obstruction.
   (mm): $2.64/c \times Lm$
   C: diameter of the catheter measured on cine.
   2.64: actual diameter of the no. 8 French catheter.
   Lm: measured length of systolic obstruction.

Corresponding to the degree of systolic compression patients were divided into 3 groups.

Group 1: 1(%)−30(%) systolic compression. (Mild)
Group 2: 31(%)−50(%) systolic compression. (Moderate)
Group 3: 51(%)−100(%) systolic compression. (Significant)

In those patients demonstrating isolated coronary bridges, exercise stress tests were performed using Tc-99m MIBI myocardial perfusion scintigraphy (SPECT image) with dose of 10 mCi during rest and 20 mCi during stress to assess the presence of exercise-induced ischemia.

RESULTS

Of the 2,547 patients studied, 511 (20 %) had no evidence of angiographically significant coronary artery disease. Twenty-six of these patients were noted to have myocardial bridging, the total incidence of myocardial bridge was 1 %. Thirty-one patients with myocardial bridging were excluded from the study because of significant accompanying coronary artery disease. Fifiy percent (13 pts) of patients without obstruction had been hospitalized at least once for a cardiac complaint, 61 % (16 pts) had been symptomatic for less than one year, and 69% (18 pts) were previously diagnosed as having typical angina pectoris. The resting electrocardiogram was normal in 17 patients (65%) whereas 7 patients (27%) had non-specific ST-T wave changes and 2 patients had minor conduction abnormalities. Only 12% of the patients with isolated myocardial bridges were women.

Of the 26 patients with isolated myocardial bridges, 4 (15%) had mild systolic coronary compression; 9 (35%) had moderate systolic compression and 13 patients (50%) had significant compression. Only two patients demonstrated ischemia by Tc-99m MIBI myocardial perfusion scintigraphy and both of these patients were in the group with severe systolic compression. Both patients were men. However, in one patient the reperfusion defect was not in the distribution of the culprit artery and was therefore considered to be a false positive.

DISCUSSION

The incidence and pathophysiologic significance of myocardial bridges has been

Table 1 Diagnosis in patients with isolate myocardial bridge.

DIAGNOSIS	Group Ⅰ n = 4	Group Ⅱ n = 9	Group Ⅲ n = 13	Total n = 26
Angina pectoris	3	6	9	18
Myocard infarctus	—	—	—	—
Syncope/Presyncope	1	—	—	1
Arrhythmia	—	1	2	3
Atypic chest pain and symptoms	1	2	1	4
Conduction abnormality	—	1	1	2
Presence of exercise induced Ischemia detected by 99mTc-Sesta MIBI	—	—	2	2

Table 2 Mean percentage and length of systolic compression due to myocardial bridging.

*Group Degree of systolic compression (%)	Mean systolic compression (%)	Mean length of systolic compression (mm)
1 (1–30)	28.7 ± 1.25	10.7 ± 1.93
2 (31–50)	42.2 ± 1.21	16.2 ± 1.89
3 (51–100)	63.1 ± 2.10	13.6 ± 1.63

* Corresponding to the degree of systolic compression, patients were divided into 3 groups.

controversial since their initial description by Grainicianu in the 1920's. Not surprisingly, pathologic evaluation of autopsy specimens has revealed a relatively substantial number of myocardial bridges [2, 5, 16]. By contrast, numerous, albeit small, angiography studies have found a much lower percentage of patients with detectable myocardial bridges [9, 15]. This disparity is likely due to the lack of sensitivity of the angiogram in detecting small changes in the caliber of coronary arteries. However, not only has the incidence of myocardial bridges been of concern, but there has also been considerable controversy regarding their functional significance. Indeed, several investigators have proposed that the constriction of the artery might well provide benefits by decreasing the diastolic shear stress to which the artery is exposed [8, 13]. Alternatively, several case reports have noted the association of myocardial bridges and tachyrythmias, episodes of unstable angina, myocardial infarction, and sudden death [1, 3, 4, 11, 14].

However, the results of the present study would suggest that myocardial bridges are a relatively benign phenomenon. Only 1% of over 2500 patients demonstrated isolated bridges and within this population only a single patient had a positive stress test that could be attributed to the bridge as a culprit lesion. Therefore, in only 1 of 2547 patients could their chest pain syndrome be attributable to a myocardial bridge. While our results cannot preclude the possibility that myocardial bridges might have contributed to the disease that was identified in the population of patients with angiographically demonstrable coronary artery disease, Julliere and colleagues studied the incidence and long-term prognosis on 28 patients with of isolated MB [10]. They reported that the long-term prognosis isolated MBs of the left anterior descending coronary artery is good and is independent of the severity of systolic narrowing of internal lumen diameter. Although each patient must be viewed separately, the probability that an anginal syndrome in a given patient is due to a myocardial bridge is quite low.

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