Original article:

Myocardial bridge – a not so rare finding in patients undergoing coronary angiography for chest pain

[1]Dr. Pradeep Meena*, [2]Dr. Deepak Maheshwari, [3]Dr. Sunil Sharma, [4]Dr. Harneesh Randhawa, [5]Dr. Pawan Goel, [6]Dr. Randheer pal

[1,3,4,5,6] Senior resident, [2] Associate professor, Department of cardiology,

Sawai Man Singh Medical College, Jaipur, Rajasthan, India

*Corresponding author: email : drpkmeenacardio@gmail.com

Abstract:

Background: Muscle fibers overlying the intramyocardial segment of an epicardial coronary artery are termed myocardial bridge. The aim of this study was to determine the frequency, angiographic characteristics, anatomical aspects, clinical manifestations and possible associations of myocardial bridges in a large urban Indian population of adults undergoing coronary angiography in our centre (Sawai Man Singh Medical College, Jaipur, Rajasthan, India).

Methods: The angiographic data of 3275 adult patients undergoing coronary angiography were retrospectively analysed for the diagnosis of myocardial bridge. Quantitative coronary angiography was used for analysis.

Results: Myocardial bridge was present in 42 (1.28%) of the 3275 coronary angiographies. The location of the bridge was in the left anterior descending coronary artery in 40 cases (95.23%), and the left circumflex coronary artery in 2 cases (4.76%). Of the 42 patients with myocardial bridges 31 (73.8%) had associated significant coronary artery disease. Remaining 11 (26.19%) patients presented with isolated bridges. Of the 11 patients of isolated myocardial bridges, 3 (27.27%) patients presented with acute myocardial infarction. The mean length of bridge was 19.07±8.86 mm and mean percentage of systolic obliteration by the bridge was 54.40±19.67%.

Conclusion: Chest pain was the common reason for angiography in patients with myocardial bridge. The incidence of myocardial bridge may vary according to population. Myocardial bridge is more frequently found in the middle segment of the left anterior descending coronary artery. Myocardial bridging can accelerate atherosclerosis and precipitate acute myocardial infarction.

Key words: Angiography, Myocardial bridge

Introduction

Muscle overlying the intramyocardial segment of an epicardial coronary artery, first mentioned in 1737 by Reyman and described angiographically in 1960 is termed myocardial bridge (MB)[1,2] .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. Myocardial bridge is clinically significant when associated with regional hemodynamic alterations, and studies have shown that such instances of myocardial bridging are linked to clinical complications that include ischemia, acute coronary syndrome, coronary spasm, arrhythmia, and sudden death, although in the vast majority of cases, myocardial bridging remains clinically silent [3–6]. There is a wide discrepancy in the reported prevalence of myocardial bridging between autopsy findings (average 33%, range 15% to 85%)[1,2,7–11] and those of

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conventional angiography (average 5%, range 0.5% to 16%) [2,3,11–16] . Detection of MB is essential while investigating cardiac ailments since it had been found to be associated with ischemic heart disease and cardiomyopathy. Specific medical and surgical treatment as is possible in myocardial bridge with clinical symptoms, its study becomes more important.Our goal was to determine the frequency, angiographic characteristics, anatomical aspects, clinical manifestations and possible associations of myocardial bridges in a large urban Indian population of adults undergoing coronary angiography in our centre for evaluation of anginal symptoms.

Material & Methods

We retrospectively evaluated 3275 consecutive patients seen in our medical center between January 2013 and september 2013 admitted with a diagnosis of possible coronary artery disease (CAD) requiring diagnostic coronary arteriography.

All patients underwent an echocardiogram and routine pre-procedure care prior to a coronary angiography using standard procedures. Each cineangiogram was reviewed retrospectively by at least two qualified cardiologists who measured the coronary lumen diameters. Arteriographic quant-ification of systolic lumen compression was performed using a programmable digital caliper to measure the systolic lumen diameter reduction and the length as previously described [18]. 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%.

Corresponding to the degree of systolic compression patients were divided into 3 groups. Group 1 comprised patients with < 50% systolic compression (Mild) of the epicardial vessel due to the myocardial bridge. Patients with 50%-70% systolic compression (Moderate) were allotted Group 2 whereas those with >70% systolic compression (Significant) were assigned to Group 3.

Informed consent was obtained from all the patients before enrollment into this study. The procedures followed were in accordance with ethical standards of the S.M.S. Medical college Ethics Committee.

Observations & results

Of the 3275 patients studied, 507 (15.48%) had no evidence of angiographically significant coronary artery disease. Myocardial bridge was present in 42 of the 3275 coronary angiographies reviewed, constituting an incidence of 1.28%. Bridging occurred in 7 of 732 females (0.95%) and 35 of 2543 males (1.37%). Selective demographic and angiographic characteristics are shown in Tables 1 and 2. Of the 42 patients with angiograms judged adequate for the diagnosis of myocardial bridging, 35 were males and 7 were females. The mean age ± standard deviation was 54.11% ± 10.04 (range: 26-75 years). As shown in Table 1, 3 (7.14%) patients had diabetes mellitus, 7 (16.6%) had systemic hypertension, 16 (38.09%) had dyslipidemia, 5 (11.9%) had family history of CAD and 24 (57.14%) were active smokers at the time when coronary angiography was performed.

Table 1. Demographic characteristic (n=42)	
Age (yrs)	54.11 ± 10.04
Male	35 (83.3 %)
Hypertension	7 (16.6 %)
Diabetes	3 (7.14 %)
Dyslipidemia	16 (38.09 %)
Smoking	24 (57.14 %)
Family history of CAD	5 (11.9 %)
Family history of sudden cardiac death	0

Each of these patients with myocardial bridge was taken up for angiography because of various symptoms which included complaints of chest pain alone, chest pain with palpitations, dyspnoea or syncope. Of the 42 myocardial bridge patients, 29 (69.04%) had history of typical chest pain and atypical presentation was seen in 13 (30.95%) patients.

31 (73.8%) of these patients with myocardial bridging had significant accompanying atherosc-lerotic CAD, whereas 11 (26.19%) patients had no evidence of angiographically significant CAD. 31 patients of atherosclerotic occlusive disease, 21 (67.74%) had atherosclerosis in the proximal region, 7 (22.58%) had atherosclerosis in the distal region and 3 (9.67%) had atherosclerosis in both proximal and distal regions to myocardial bridge. Of the 31 patients of myocardial bridges with significant CAD, 20 (64.51%) patients had single vessel disease, 9 (29.03%) patients had double vessel disease and 2 (6.45%) patients had triple vessel disease. Right coronary artery (RCA) was the dominant vessel in most of the bridging vessels, seen in 35 (83.33%) patients. (Table-2)

Table 2. Echo and angiographic charateristic (n=42)	
Left ventricular hypertrophy	7 (16.1%)
LV systolic dysfunction	14 (33.33%)
LV diastolic dysfunction	19 (45.23%)
LV ejection fraction (%)	50.97% ± 9.57
Dominance	
Right coronary artery	35 (83.33%)
Left coronary artery	7 (16.66 %)
Isolated myocardial bridge	11 (26.19%)
Myocardial bridge with accompanying significant CAD	31 (73.8%)
Myocardial bridge + 1 vessel disease	20 (64.51%)
Myocardial bridge + 2 vessel disease	9 (29.03%)
Myocardial bridge + 3 vessel disease	2 (6.45%)

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Mean Length of myocardial bridge (mm)	19.07 ± 8.86
Mean systolic lumen compression (%)	54.40% ± 19.67

As shown in table-3, Of the 11 patients of isolated myocardial bridges, 6 (54.54%) patients presented with angina pectoris, 3 (27.27%) patients presented with myocardial infarction, 1 (9.09%) patient presented with syncope and 1 (9.09%) patient presented with atypical chest pain. Of these 11 patients, the resting electrocardiogram was normal in 3 (27.27%) patients whereas other 8 (72.72%) patients had ST-T wave changes and minor conduction abnormalities. Treadmill exercise test was positive only in one patient.

Table 3. Presenting abnormalities in patients with isolated myocardial bridge (n=11)

Presenting abnormalities	Group - 1 (n=3)	Group - 2 (n=4)	Group – 3 (n=4)	Total (n=11)
Angina	1	2	3	6
Myocardial infarction	0	2	1	3
Syncope	1	0	0	1
Arrhythmia	0	0	0	0
Atypical chest pain	1	0	0	1
Conduction abnormalities	0	1	1	2
ECG abnormalities	1	3	4	8
Treadmill test positive	0	1	0	1

Corresponding to the degree of systolic compression, 19 (45.23%) patients fall into group 1, 15 (35.71%) patients in group 2, and 8 (19.04%) patients in group 3. In group 1 the mean systolic lumen compression was 36.42% ± 6.91 and mean length of systolic lumen compression was 14.5 ± 6.63 mm, whereas in group 2 and group 3 the mean systolic lumen compression was 60.8% ± 5.42 and 85.12% ± 6.35 and mean length of systolic lumen compression was 20.0 ± 8.04 mm and 28.0 ± 8.34 mm respectively (Table-4). Overall mean length of myocardial bridge was 19.07 ± 8.86 mm and mean systolic lumen compression was 54.40% ± 19.67.

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Table 4. Mean percentage and length of systolic compression due to myocardial bridge		
Degree of systolic compression	**Mean systolic lumen compression (%)**	**Mean length of systolic compression (mm)**
Group – 1 (n=19)	36.42 ± 6.91	14.5 ± 6.63
Group – 2 (n=15)	60.8 ± 5.42	20.0 ± 8.04
Group – 3 (n=8)	85.12 ± 6.35	28.0 ± 8.34

The location of the myocardial bridge was more common in left anterior descending (LAD) coronary artery in 40 cases (95.23%), whereas left circumflex (LCX) coronary artery involved only in 2 cases (4.76%). No bridge was found in the right coronary artery system in our study. The middle one third portion of the LAD coronary artery was the most common site (27 of 40 cases, 67.5%). Involvement of the distal and proximal one third of LAD coronary artery was observed in 12 (30%) and 1 (2.5%) cases respectively.

The length of myocardial bridge ranged from 1 cm to a diffuse involvement of more than one third of the artery. The greater length of the myocardial bridge covered the mid portion and the beginning of distal portion of the LAD coronary artery.

Discussion

The incidence of myocardial bridge has been reported to be 0.5% to 7.5% in various angiographic studies (Table-5). A total of 3275 angiograms reviewed in this study, of which 42 (an incidence of 1.28%) cases had myocardial bridging. This is inconsistent with previous reports of myocardial bridging.

Table-5. The incidence and site of myocardial bridges in angiographic studies				
Previous Studies	Total number of angiograms reviewed	Patients with myocardial bridge	Incidence	Site
Noble et al, 1976	5250	27	0.5 %	LAD
Ishimori et al, 1977	313	5	1.6 %	LAD
Greenspan et al, 1980	1600	14	0.9 %	LAD
Irvin, 1982	465	35	7.5 %	LAD
Angelini et al, 1983	1100	60	5.5 %	LAD + RCA
Mavi et al, 2008	7200	29	0.4 %	LAD + LCX
Our study, 2013	3275	42	1.28 %	LAD + LCX

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The angiographic incidence of myocardial bridge reported by Irvin et al[19] was higher than that found in previous angiographic studies. He explained the higher prevalence in his study as follows: (1) The arteriograms were retrospectively reviewed for the specific purpose of assessing the frequency of myocardial bridges (2) No percentage of systolic narrowing was specified for the designation of myocardial bridge in his series (3) The technologic advances in cineangiography, which had occurred in the past few years, have allowed more precise delineation of the arterial diameters. Even though our study was similar to the one done by Irvin et al[19] the incidence of myocardial bridge in our study was 1.28%, i.e. much lower than that reported by Irvin et al[19].

Soran et al[16] reported that the frequency of myocardial bridges in 2547 Turkish patients undergoing coronary angiography was 1%. Their incidence was similar to our study because their consensus in the degree of systolic compression was 1% to 100%. When the degree of systolic compression was 50% or more, the frequency of myocardial bridges was 0.5% in their study and 0.7% in our study.

Of the 42 cases with myocardial bridging 35 patients (83.33%) had right dominant coronary system. There are pathological reports that myocardial bridges are more common with a left coronary dominant system. However there was no such relation observed in the current study.

In our study, 40 patients (95.23%) had myocardial bridge of the LAD coronary artery and 2 (4.76%) patients had myocardial bridge of the LCX coronary artery. Myocardial bridges are usually found over the LAD coronary artery; they are very rarely found over the RCA or the LCX coronary artery. Angelini et al[3] reported that out of 61 patients with myocardial bridge, only 1 had an additional myocardial bridge of the posterior descending branch of the RCA. Myocardial bridges over the LCX coronary artery are also rare in angiographic studies. A case was reported by Okmen et al[20] in an angiographic study. Necropsy reports have also shown that myocardial bridges are more frequent in the proximal LAD artery, but have been noted to involve all of the major epicardial branches [21, 22].The most frequent site of myocardial bridge is found in the middle segment of the LAD coronary artery. [3,19, 22] Our finding is consistent with the previous reports. Out of 40 patients with myocardial bridge of the LAD coronary artery, 27 (67.5%) patients had myocardial bridge in middle segment, while 1 (2.5%) and 12 (30%) patients had in proximal and distal segment of the LAD coronary artery respectively.

There are multiple reports[17] of accelerated atherosclerosis in segments of coronary artery proximal and distal to a myocardial bridge and a surprising absence of the same in the segment underlying the bridge. In our study, out of 42 patients with myocardial bridge, 31 (73.8%) had atherosclerosis in one or more coronary artery. Out of 31 patients with atherosclerosis, 21 (67.74%) had atherosclerosis in the proximal to the myocardial bridge region while 7 (22.58%) had atherosclerosis in the distal to the myocardial bridge region and 3 (9.69%) had atherosclerosis in the proximal and distal regions to the myocardial bridge. We have not found atherosclerotic plaques in the territory of the bridge. The myocardial bundles contraction, around intramyocardial coronary arteries generate a tissue pressure close to or even greater than the intravascular blood pressure, especially in systole. This significantly reduces mural stress and therefore

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plays a key role in protecting the intramyocardial portion of the arteries from arteriosclerotic lesions. The pressure and the disturbance of blood flow and high wall stress proximal to the myocardial bridge are the main contributors to the development of atherosclerosis in the proximal segment.

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 association[24] or no association[25]. In our study, the degree of systolic compression was between 20 and 95 percent. We have found a trend suggestive of an association between the degree of vessel compression and the presenting symptoms in patients with isolated myocardial bridge. Patients with isolated myocardial bridge belonging to group's 2 and 3 were higher in number, having more typical presentation, angina, MI and electrocardiographic abnormalities as compare to group 1. Whether this association is true and of any statistical significance, needs to be determined by a bigger study with higher number of cases.

Ischemia induced by myocardial bridging is related to several reasons. In addition to systolic compression and delayed diastolic relaxation, vasospasm of coronary artery may be another mechanism. The considerable delay in blood flow and reduced distal coronary pressure are presumed to impair coronary vasodilator reserve, which may induce ischemia in patients with myocardial bridging. Another reason for coronary ischemia may be related to coronary spasm, which is stimulated by endothelial dysfunction of the coronary artery. Coronary angina even myocardial infarction may occur in this situation.

Even though considered as a relatively benign condition, in the current study, 27.27% patients with isolated bridges presented with acute myocardial infarction. Whether such patients should undergo PCI is a matter of debate. Of the 31 patients with significant coronary disease, 64.51% patients had single vessel disease and they had the culprit lesion and myocardial bridge seen in the same vessel. Whether myocardial bridging predisposed for coronary disease or not is difficult to prove.

The results of this study need to be confirmed by further studies with larger populations. Moreover, as this study was performed retrospectively, it was not possible to match the patients in the angiographic performance and medical options.

Conclusions

The purpose of this report is neither to assess the pathophysiologic significance of the bridging phenomenon nor to provide an in-depth assessment of the subsequent course of these patients. The data presented here indicate that angiographically demonstrable myocardial bridging is not a rare occurrence. Myocardial bridging can accelerate atherosclerosis and precipitate acute MI. Myocardial bridging must be considered especially in patients at low risk for coronary atherosclerosis but with angina like chest pain or established myocardial ischemia. Medical management is the main stay and requires more awareness. PCI/Surgical management is still a matter of debate. It may be considered if there are recurrent events despite optimal medical therapy.

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