DOI: 10.14260/jemds/2015/1613

ORIGINAL ARTICLE

PREVALENCE OF MYOCARDIAL BRIDGING BY CORONARY ANGIOGRAM: ANALYSIS AND SIGNIFICANCE

Prabhas Ranjan Tripathy[1], Debasish Das[2], Divia Paul A[3]

HOW TO CITE THIS ARTICLE:
Prabhas Ranjan Tripathy, Debasish Das, Divia Paul A. "Prevalence of Myocardial Bridging by Coronary Angiogram: Analysis and Significance". Journal of Evolution of Medical and Dental Sciences 2015; Vol. 4, Issue 64, August 10; Page: 11202-11208, DOI: 10.14260/jemds/2015/1613

ABSTRACT: Out of nine hundred and eighty seven (987) coronary angiogram reports, in the present study we analysed the prevalence and distribution of bridging segments. The co-relation of these tunnelled segments with diseased and non-diseased coronary arteries and cardiac dominance were also assessed. Myocardial bridging was exclusively confined to LAD predominantly in the midsegmentG of nondiseased arteries in right dominant circulation. Although bridging provides an atheroprotective milieu, it even harboured atherosclerosis proximal to it. Significantly altering coronary macro and micromechanics it plays a significant role behind genesis of coronary ischemia and its management.

KEYWORDS: Anatomy, Artery, Bridge, Angiogram, Cardiology, Myocardium.

INTRODUCTION: Muscle overlying one segment of an epicardial coronary artery, first mentioned by Reyman in 1737[1] and Black in 1805,[2,3]is termed a myocardial bridge and the artery running within the myocardium is called a tunnelled artery. It is characterized by systolic compression of the tunnelled segment, artery vanishing in systolic phase and appearing in diastolic phase in coronary angiogram[4,5] which remains clinically silent in the vast majority of cases.

The surrounding myocardium appears to be a key factor in generating a unique atheroprotective microenvironment within bridges,[6] although bridges get atheroma. The first post-mortem examination of myocardial bridge was performed by Geiringer in 1951[7] and it was followed by the first radiological description by Portman and Ingrid in 1960.[8]

MATERIALS AND METHODS: Based on the coronary angiogram reports of nine hundred and eighty seven (987) patients, who were evaluated under the Cardiology outpatient department of KIMS Hospital and AIIMS, Bhubaneswar and Non- Communicable Disease (NCD) clinic of AIIMS, Bhubaneswar from the time period of 2010 April to 2014April, the following reports were obtained from the Cardiac Cath Laboratory. Reports were analysed statistically in percentages and p values were obtained.

Angiogram reports were analysed as follows:

- Prevalence of bridging.
- Distribution of bridging.
- Segments involved in bridging.
- Cardiac dominance.
- Distribution of bridging segment in diseased and non-diseased coronaries.
- Normal and diseased coronary among bridging.
- Prevalence of bridging.

DOI: 10.14260/jemds/2015/1613

The bridging segments were seen in thirty five (35) cases among nine hundred and eighty seven (987) angiogram reports. This was reaching up to a percentage of 3.5%. {Table no: 1}

Distribution of Bridging in Coronary Artery: Myocardial bridging was observed only in left anterior descending branch of left coronary artery (LAD) with a percentage of 3.5%.{Table no: 2}. No bridging pattern was observed in Diagonal, Left circumflex, Ramus, Obtuse Marginal, Right Coronary (RCA) arteries.

Segments Involved in Bridging: Bridging segment pattern commonly divided into proximal, middle and distal involvement. In the present study no proximal segment involvement was evident. Twenty five (25) cases exhibited middle segment involvement and ten (10) cases showed distal segment involvement. The percentages of mid and distal segments were 71.4% and 28.6% respectively among 35 bridged segments. {Table no: 3}

Cardiac Dominance: Right, Left and Co-dominance among the coronary arteries were analysed. There were eight hundred and thirteen (813) cases of right dominance (82.5%).Left dominance was there in 131(13.3%) cases. Co-dominance was seen in 44(4.4%) cases.{Table no: 4}.When the cardiac dominance pattern were correlated to bridging segments 29 segments were seen in right dominance, 5 bridging segments falls under left dominance and 1 under codominant pattern demonstrated in Table 4 with a correlation coefficient of 99.96% (Table 7).

Distribution of Bridging Segment in Diseased and Non-diseased Artery: Diseased segments were observed in five hundred and forty three arteries (543), out of whom 6(1%) cases had myocardial bridge. Four hundred and forty four (444) cases falls under the category of non-diseased arteries. Twenty nine (29) segments were bridged under non–diseased arteries {Table no: 5, 9}.On analysing the disease pattern of bridges, fifteen(15) cases showed normal coronary arteries and twenty(20) cases showed diseased coronary arteries. It was only three (3) out of twenty cases in LAD was diseased {Table no: 6}.

ANGIOGRAM REPORTS	BRIDGING SEGMENTS N = 35 (n %)
987	35 (3.5%)

Table 1: Prevalence of bridging

ARTERY n=(987)	LAD	Diagonal Branch	LCX	Ramus Branch	Obtuse Marginal	RCX
NUMBER	35	0	0	0	0	0
PERCENTAGE (100%)	3.5%	0	0	0	0	0

Table 2: Distribution of bridging

No bridging pattern was observed in Diagonal branch, Left circumflex artery, Ramus branch, Obtuse Marginal, RCX artery.

DOI: 10.14260/jemds/2015/1613

SEGMENT	NUMBER N = 35 (n %)
Proximal segment	0(0%)
Mid segment	25 (71.4%)
Distal segment	10 (28.6%)

Table 3: Segments involved in bridging (out of 35)

DATA	NO. OF ARTERIES N = 987 (n %)	NO. OF BRIDGES N = 35 (n %)
Right Dominance	813 (82.5%)	29 (3.6%)
Left Dominance	131 (13.3%)	5 (3.8%)
Co-Dominance	43(4.4%)	1 (2.3%)

Table 4: Cardiac dominance (out of 987)

DATA	NO. OF ARTERIES	NO. OF BRIDGES N = 35 (n %)
Diseased	543	6(1.1%)
Non-Diseased	444	29 (6.5%)
TOTAL	**987**	**35 (3.5%)**

Table 5: Distribution of bridging segment in diseased and non-diseased artery (out of 987)

Data	NUMBER N = 35 (n %)
Normal coronary	15 (43%)
Diseased coronary	20 (57%)
Lad bridging segment in diseased coronary(out of 20)	3 (15%)
Non-lad bridging segment in diseased coronary(out of 20)	17 (85%)

Table 6: Normal and diseased coronary among bridging (out of 35)

Data	No. of Arteries	No. of Bridges	Correlation Coefficient 99.96%
Right Dominence	813	29	
Left Dominence	131	5	
Co-Dominence	43	1	
TOTAL	**987**	**35**	

Table 7

DOI: 10.14260/jemds/2015/1613

Data	No. of Arteries	No. of Bridges	Correlation Coefficient 17.62%
Diseased	543	6	
Non-Diseased	444	29	
TOTAL	**987**	**35**	

Table 8

	With Bridges	Without Bridges	Row Total	x_2=21.03 DF=1 p=0.000005
Diseased	6	537	543	
Non-Diseased	29	415	444	
Column Total	**35**	**952**	**987**	

Table 9

DISCUSSION: Iskandrian and others [9-13] in their studies of myocardial bridging mentioned that among patients undergoing coronary angiography, the reported prevalence of myocardial bridging is 1.7 percent (range 0.5 to 16 percent), which is almost always confined to the left anterior descending artery (LAD) where as the present study reveals it as 3.5% only confined to LAD as well. Mookadam et al[14] in coronary angiography of 14, 416 patients reported myocardial bridging in 1.57% of cases. Pereira et.al[15] showed the phenomenon of systolic constriction of the LAD diagnostic of myocardial bridge in 123 cases out of 3375 coronary angiographies and the frequency of myocardial bridge on cineangio cardiography performed in this period was 3.6%.

In contrast to this, Loukas M et.al [16] examined the relationship of myocardial bridge to coronary artery dominance in two hundred formalin-fixed human hearts where myocardial bridges were found in 69 (34.5%) cases. One bridge was found in 59 of these hearts and multiple bridges were observed in ten (eight with double bridges and two with triple bridges).Bridges were most often found in LAD (35 hearts). Bridges were also found in diagonals (14), OMs (Five) and over the inferior interventricular branch of the left coronary artery (Six). Bridges were also found over the right coronary artery (15 hearts), over the right marginal branch (Four) and over PDA (two).

Journel et al[17] published an article of on a study aimed to assess the prevalence and characteristics of myocardial bridging in a total of 277 patients (mean age 60+/-11 years) who underwent multislice computed tomography and coronary angiography(MSCT-CA).Segments proximal and distal to the bridging were assessed for atherosclerotic plaque, as were the remaining coronary segments. Myocardial bridging was present in 82 patients (30%, mean age 59+/-12 and frequently localised in the mid-distal segment of the left anterior descending artery (95%).

In the present study, when compared to the above mentioned study, the findings were equivocal as mid and distal segment were only involved in bridging pattern with a percentage involvement of 71.4 % and 28.6% respectively. Mid segment involvement was more compared to the distal segment.

Mohimuddinn et.al.[18] in their studies among 107 patients for the relationship between myocardial bridge type and proximal stenosis in hypertensive patients. In this study, superficial bridges were the most common form of myocardial bridging (64.4%) with deep bridge in rest 35.55% and the most common artery involved was left anterior descending artery. Among superficial bridges most common location of the bridge in the left anterior descending was in the middle segment (>80%)

of the artery, while the second most common location was the proximal part of the artery. Among deep bridges more than 90% of the bridge was found in the middle segment, while the remainder was in the proximal segment.

Comparing this, the present study was also having 71.4% of mid segments exhibiting the bridging pattern but there were no proximal segment in which bridging pattern was seen.

Loukas et al[19] analysed the relationship of myocardial bridges to coronary artery dominance among two hundred formalin-fixed human hearts. The presence of bridges appeared to be related to coronary dominance, especially in the left coronary circulation. Forty-six (66.6%) hearts with bridges were left dominant. Seventeen hearts (24.6%) were right dominant. The remaining six hearts were co-dominant.

Comparing this with the present study, the bridging segments percentage with right dominance are more than the left dominance percentage. Co-dominance percentage is less compared to right dominance and left dominance bridging segments percentages.

Jeremias et al[20] analyzed 69 patients with myocardial bridges, using intravascular ultrasound found that there was a high incidence of atherosclerosis at the segments proximal to the bridges but no plaque was found within and distally to the bridges. Herrmann et al[21] in their studies of myocardial bridging demonstrated its association with alteration in coronary vasoreactivity.

Robicsek et al[22] studied the phenomenon that coronary arteries which course entirely intramyocardially remain immune to atherosclerosis in 250 patients undergoing coronary bypass surgery. Of these out of 26 patients with intramyocardial bridges, 24 had no atherosclerosis in the intramyocardial bridging segment, one had a calcified plaque, and one had a recanalized thrombus, there was no apparent atherosclerosis in the intramyocardial segments. It is therefore concluded that the freedom from atherosclerosis of the intramyocardial coronary arteries is due to the lack of mural stress, i.e. the lower or absent transmural pressure gradient.

Yiannis et al.[23] found myocardial bridges are free from atherosclerosis. Overview of the underlying mechanisms suggested that the surrounding myocardium appears to be a key factor by generating a unique atheroprotective hemodynamic microenvironment beneath bridges and a highly atherogenic milieu at their proximal edges. The main components of this environment include low tensile stress and high shear stress. Reduced coronary wall motion due to external support of the surrounding myocardium may also play a role.

Comparing the above mentioned studies[20-23] with the present study 1.1% were bridged in diseased artery was and 6.5% arteries were bridged under non–diseased arteries. Left anterior descending artery (LAD) had all the thirty five bridging segments where as only three (3) cases out of twenty diseased coronaries had myocardial bridge underneath which correlates the present studies.

CONCLUSION: Myocardial bridging although being a known etiology behind coronary ischemia, plays a critical role in management. Almost always confined to LAD, other arteries involvement are only of historical autopsy literature. Although tunnelling provides an atheroprotective milieu, proximal to bridging segment atherosclerosis becomes evident. Mid segment involvement is commonest although no segment behaves as exception. Bridging alters the micro and macro coronary mechanics inviting and propelling atherosclerosis at the same time; a double edged sword to jeopardise the healthy myocardium, so we should be careful and cautious about it.

REFERENCES:

1. Reyman HC. Disertatis de vasiscordispropiis. Bibl Anat 1737; 2:366.
2. Black S. A case of angina pectoris with dissection. Memoirs Med SocLond.1805; 6:41.
3. Myocardial Bridge: Texas Heart Institute Heart Information. Oct 2013.
4. Endo M, Lee YW, Hayashi H, et.al. Angiographic evidence of myocardial squeezing accompanying tachyarrhythmia as a possible cause of myocardialinfarction.Chest.1978; 73: 431–433.
5. Hwang JH, Ko SM, Roh HG et.al. Myocardial bridging of the left anterior descending coronary artery: depiction rate and morphologic features by dual-source CT coronary angiography. Korean J Radiol.2010; 11:514–521.
6. Yiannis S Chatzizisis, George D Giannoglou. Myocardial bridges are free from atherosclerosis: Overview of the underlying mechanisms. Can J Cardiol. Apr 2009; 25(4): 219–222.
7. Geiringer E. The mural coronary. Am Heart J. 1951; 41:359–368.
8. Portman W, Ingrid J. Intramural coronary vessels in the angiogram. Fortschr Geb Rontgenstr Nuklearmed. 1960; 92:129–133.
9. Paul Sorajja and Ami E Iskandrian .Myocardial bridging of the coronary arteries J. Upto date. Dec 3, 2012.
10. Möhlenkamp S, Hort W, GeJ et.al. Update on myocardial bridging. Circulation 2002; 106:2616.
11. Alegria JR, Herrmann J, Holmes DR Jr, et al. myocardial bridging. Eur Heart J 2005; 26:1159.
12. Iskandrian AE, Nallamothu N, Heo J. Nonatherosclerotic causes of myocardial ischemia. J NuclCardiol 1996; 3:428.
13. La Grutta L, Runza G, Lo Re G, et al. Prevalence of myocardial bridging and correlation with coronary atherosclerosis studied with 64-slice CT coronary angiography. Radiol Med 2009; 114:1024.
14. Mookadam F, Green J, Holmes D et. al. Clinical relevance of myocardial bridging severity: single center experience. Eur J Clin Invest. 2009 Feb; 39(2):110-5.
15. Pereira AB, Castro DS, Menegotto ET et.al. Myocardial bridging: therapeutic and clinical development. Arq Bras Cardiol. 2010; 94(2):175-81.
16. Loukas M, Curry B, Bowers M, et.al. The relationship of myocardial bridges to coronary artery dominance in the adult human heart.J Anat. 2006 Jul; 209(1):43-50.
17. Journel L, Midiri M. Multislice CT angiographic evaluation of myocardial bridging, La Radiologia Medica.2009, 114(7):1024-1036.
18. Gouse Mohimuddin, Xindao Yin, Hui Xu, et. al. The relationship between myocardial bridge type and proximal stenosis in hypertensive patients. Korean J Radiol. 2014; 11(1): 22-26.
19. Loukas M, Curry B, Bowers M, et. al .The relationship of myocardial bridges to coronary artery dominance in the adult human heart.J Anat. 2006 Jul; 209(1):43-50.
20. Ge J, Jeremias A, Rupp A, et al. New signs characteristic of myocardial bridging demonstrated by intracoronary ultrasound and Doppler. Eur Heart J. 1999; 20:1707–16.
21. Herrmann J, Higano ST, LenonRJ, et.al. Myocardial bridging is associated with alteration in coronary vasoreactivity. Eur Heart J. 2004; 25:2134–42.
22. Robicsek F, Thubrikar MJ. The freedom from atherosclerosis of intramyocardial coronary arteries: Reduction of mural stress – a key factor. Eur J Cardiothorac Surg. 1994; 8:228–35.
23. Yiannis S Chatzizisis, George D Giannoglou. Myocardial bridges are free from atherosclerosis: Overview of the underlying mechanisms. Can J Cardiol. Apr 2009; 25(4): 219–222.

DOI: 10.14260/jemds/2015/1613

ORIGINAL ARTICLE

AUTHORS:

1. Prabhas Ranjan Tripathy
2. Debasish Das
3. Divia Paul A.

PARTICULARS OF CONTRIBUTORS:

1. Assistant Professor, Department of Anatomy, AIIMS, Bhubaneswar.
2. Assistant Professor, Department of Cardiology, AIIMS, Bhubaneswar.
3. Tutor, Department of Anatomy, AIIMS, Bhubaneswar.

FINANCIAL OR OTHER COMPETING INTERESTS: None

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Debasish Das,
Assistant Professor,
Department of Cardiology,
AIIMS, BBSR.
E-mail: dasdebasish54@gmail.com

Date of Submission: 10/07/2015.
Date of Peer Review: 11/07/2015.
Date of Acceptance: 04/08/2015.
Date of Publishing: 08/08/2015.