ECRONICON OPEN ACCESS

EC CARDIOLOGY
Research Article

Myocardial Bridge Analysis by 128-MDCT and its Association with Coronary Atherosclerosis in the Proximal Segment

Adel Mohamed khalifa*

Alexandria university , Egypt

***Corresponding Author**: Adel Mohamed khalifa, Alexandria university , Egypt.

Received: November 17, 2016; **Published:** November 30, 2016

Abstract

Objective: To evaluate the presence and distribution of atherosclerotic plaques in relation to myocardial bridge coronary segments and to determine the prevalence of myocardial bridges and their location and morphology using 128-MDCT.

Methods: This study included 100 patients with MB selected among 1950 patients presented to the International Cardiac Center, underwent CCTA using 128-MDCT scan From January 2015 to April 2016.Detection of the presence of Myocardial Bridge: as regard site, length, depth and degree of systolic obstruction, Coronary plaque assessment. Significance of the obtained results was judged at the 5% level.

Results: Among the 100 patients we found 30 patients having MB and proximal CAD included in Group(A), 70 patients having MB without CAD included in Group(B), In group(A), MB was in Mid LAD in 20(66.7%) patients, Distal LAD in 10(33.3%) patients the mean length of MB was 24.80± 11.93 mm the mean depth of MB was 3.81± 2.30 The mean Degree of systolic obstruction of MB was 68.87± 19.42. While in group(B), MB was in Mid LAD in 37(52.9%) patients, Distal LAD in 25 (35.7%) patients and Proximal LAD in 8 (11.4%) patients. The mean length was 24.21 ± 12.08 mm. The mean depth was 2.91 ± 1.60 ($p = 0.019$), The mean Degree of systolic obstruction was 31.07 ± 17.91 ($P < 0.001$).

Conclusion: Certain anatomic characteristics of MB, such as depth and degree of systolic obstruction, may contribute to the development of atherosclerosis.

Keywords: *Myocardial Bridge Analysis; 128-MDCT; Coronary Atherosclerosis*

Introduction

Myocardial Bridging an inborn coronary abnormality [1,2], is defined as a segment of a major epicardial coronary artery, the 'tunneled artery', that goes intramurally through the myocardium beneath the muscle bridge. It was recognized at autopsy by Reyman in 1737 [3] and first described angiographically by Portmann and Iwig in 1960 [4].

Myocardial Bridging was first described by Geiringer in 1951 [5] studied by dissection method on autopsy samples and reported an incidence of 23% with predominance of myocardial bridges on anterior interventricular artery.

Polacek in 1961[6] examined 70 hearts and reported an incidence of myocardial bridges of 85.7%.

According to text (Gray's Anatomy) The anatomical distribution of muscle bridges are classified into two as types: superficial (75% of cases) [7] or deep (25% of cases), depending on the thickness of the covering muscle layer [9]. Additionally, the superficial type can be further classified as complete or incomplete (Figure 1).

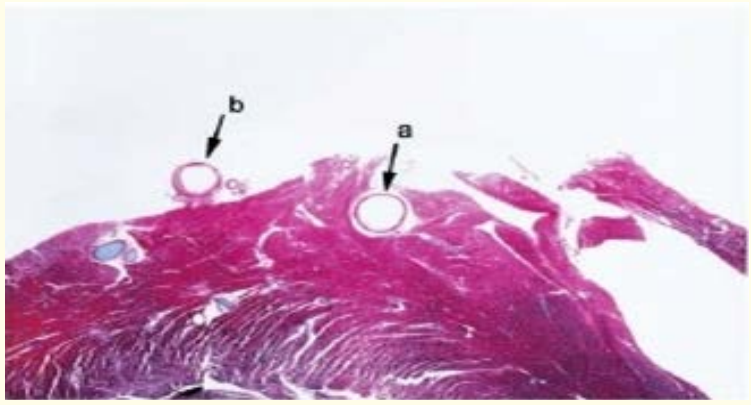

Figure 1: *Histologic cross section showing (a) a tunneled seg- ment and (b) an epicardial branch of the LAD.*

The distinction between superficial and deep muscle bridges is important in ischemia and may explain why some demonstrable muscle bridges do not cause symptoms. Showing that deep muscle bridges surround the left anterior descending coronary artery with helices of muscle fibres and speculate that this may distort or compress the adjacent artery (Figure 2) [9].

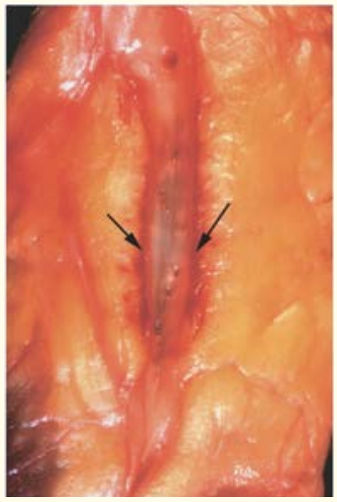

Figure 2: *Pathological specimen showing an opened coronary artery with a thin myocardial bridge (arrows) and adjacent proximal and distal epicardial segments.*

Incidence of Myocardial Bridge

The incidence of myocardial bridging has been reported between 15 and 85% in autopsy studies [10,11]. The frequency reported in angiographic studies varies from 0.5 to 16% [12,13]. On the other hand, the rate rises to 40% with the provocation test used during conventional angiography [15-16]. This gap between angiography and autopsy series has been attributed to multiple factors including the length and depth of the tunneled artery, with only deeply located coronary artery segments within the ventricular myocardium appearing to be sufficiently compressed during systole to be identified on angiography. In addition, the presence of atherosclerotic plaques proximal to myocardial bridging may cause underdiagnoses [1,14].

The mid segment of the left anterior descending artery (LAD) is the most frequent site of bridging, followed by the distal LAD (Figure 3).

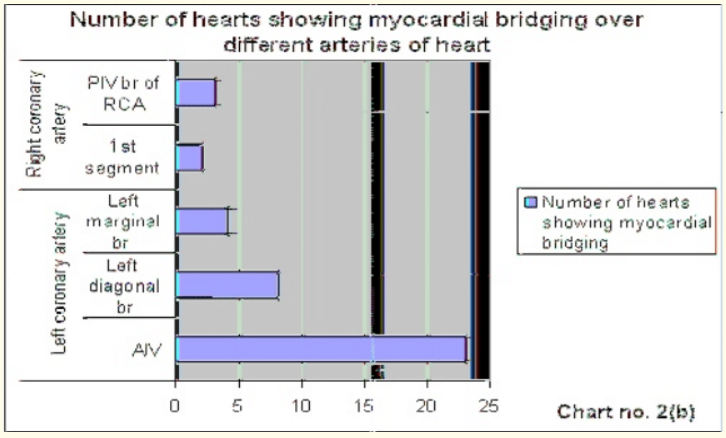

Figure 3: *Journal of the Anatomical Society of India 57.1 (2008): 14-21.*

Morphology

Myocardial bridges vary in size with a reported length ranging from 4 to 40 mm by autopsy [17,18] 8 - 50 mm by coronary CTA [19,20], and a width and depth between 1 and 4 mm by autopsy and 1 – 3 mm by coronary CTA [19,20].

Longer and deeper bridges and those that exhibit greater degrees systolic compression (0.70%) are more common in symptomatic patients [18-21].

On coronary CTA, the length and the depth of myocardial bridging are most used describe bridges. In general, a depth of bridging of ≥ 2 mm is considered deep.

Clinical Impact of Myocardial Bridge

In most patients, myocardial bridging is an incidental finding associated with an excellent survival rate of 97% at 5 years [23].

However, it is not entirely a benign entity. There have been reported associations with myocardial ischemia, [24] myocardial infarction [25-27], arrhythmia [28], and sudden death [29].

This systolic milking effect along with associated altered coronary flow dynamics within the bridged segments (increased diastolic flow velocity and average flow velocity) may result in impaired coronary flow reserve and ischemia [23,30-31].

Myocardial bridging occurs frequently in patients with hypertrophic cardiomyopathy, with a prevalence as high as 30% [32-34].

Treatment

For asymptomatic patients, no treatment is necessary. Pharmacologic therapy with B-blockers or non-dihydropyridine calcium channel blockers and antiplatelet agents with the objective of relieving symptoms and signs of myocardial ischemia and/or protecting against the risk of future coronary events [35]. Nitrates are contraindicated in patients with myocardial bridging.

For patients with severe symptoms refractory to medical treatment and recurrent clinical events, percutaneous or surgical intervention may be considered [36]. The use of stenting, however, is controversial.

The mainstay of surgical therapy is coronary artery bypass grafting to segments distal to the myocardial bridging to improve the blood flow to compromised areas, or surgical unroofing of the intramyocardial coronary segment (myotomy) [37] (Figure 4).

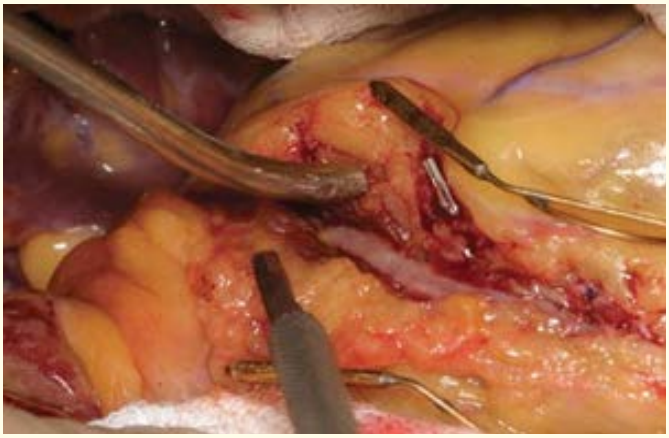

Figure 4: *Surgical unroofing of the intramural coronary with myotomy.*

Coronary Angiography

The current gold standard for diagnosing myocardial bridges is coronary angiography with the typical "milking effect" and a "step down–step up" phenomenon induced by systolic compression of the tunneled segment [38]. However, these signs provide little information on the functional impact at the myocardial level. In the presence of a proximal stenosis, myocardial bridging may only be identifiable after PTCA when higher intravascular pressures and reversed hypokinesis unmask myocardial bridging (Figure 5).

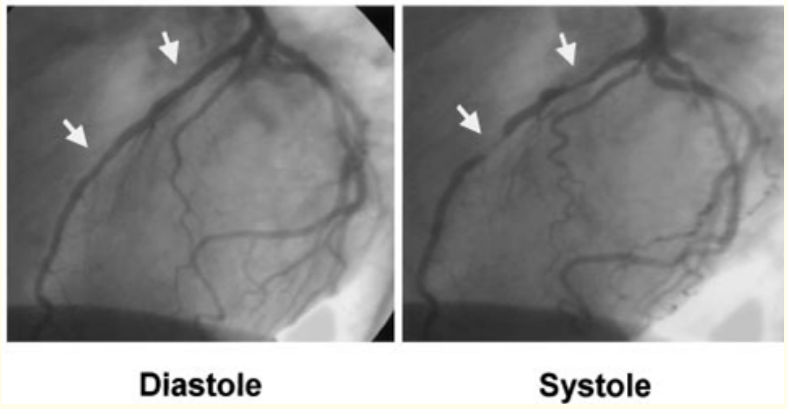

Figure 5: *Typical systolic compression (arrows) of the mid LAD at two sites in series.*

In patients with thin bridges, the milking effect may be missed and new imaging techniques and provocation tests may be required to detect a bridge.

Intracoronary Ultrasonography and Doppler Evaluation of Myocardial Bridges

The "half-moon phenomenon" is a characteristic IVUS observation [39]. It seems specific for the existence of myocardial bridging in as much as it is only found in tunneled segments but not in proximal or distal segments or in other arteries. In the presence of a half-moon phenomenon on IVUS, milking can be provoked by intracoronary provocation tests, even if the bridge was angiographically undetectable (Figure 6).

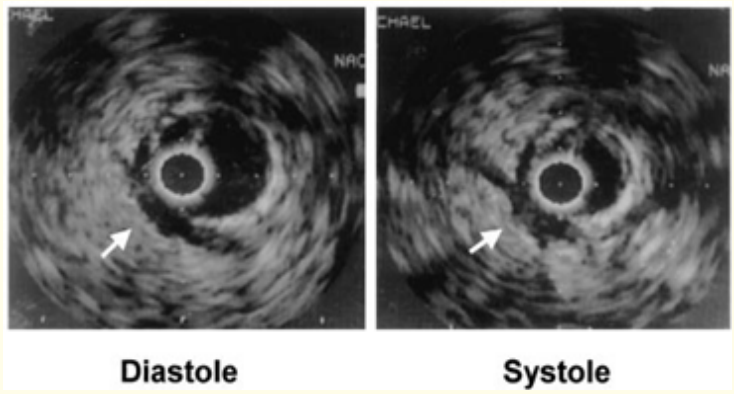

Figure 6: *IVUS-images of the myocardial bridge during diastole (left) and systole (right). A "half-moon"–like area surrounding the tunneled segment is present during the entire cardiac cycle.*

In ICD studies, pullback of the Doppler-flow wire frequently reveals a characteristic flow pattern, the "fingertip phenomenon" or "spike-and-dome pattern" (Figure 7). This flow pattern had previously been described in experimental studies and consists of a sharp acceleration of flow in early diastole followed by immediate marked deceleration and a mid-diastolic pressure plateau.

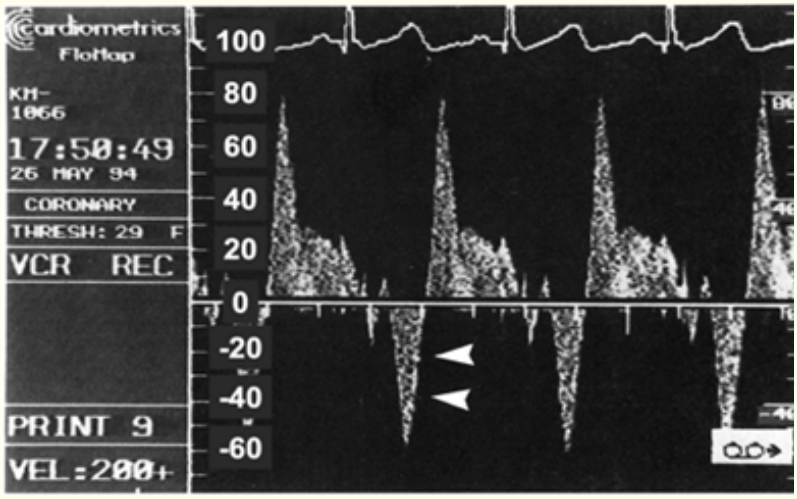

Figure 7: *ICD-images of the myocardial bridge showing retrograde flow during systole (double arrows) in the proximal segment of the bridge after nitroglycerin provocation. A typical "fingertip" phenomenon can be visualized in diastole (single arrow).*

MDCT Angiography Evaluation of Myocardial Bridge

MDCT angiography offers advantages over conventional angiography for the evaluation of myocardial bridging, because it is a noninvasive imaging modality that allows assessment of the coronary artery lumen, wall, and surrounding myocardium it yields information on the length, depth, and precise location of atherosclerosis associated with myocardial bridging. The identification of vulnerable coronary plaque.

Methods

All patients will be subjected to

- Cardiovascular risk factors including: age, sex, smoking history, presence of hypertension, dyslipidemia, diabetes mellitus and family history of CAD.

MSCT coronary angiography:

- MSCT coronary angiographic studies using 128-MDCT (Aquilion 128-Toshiba Medical system).
- Patients will be fasting for 4–6 hours. Oral B-blocker administered one hour before the scan for those with a heart rate above 65 beats/ minute.
- A bolus of 80 ml of intravenous non-ionic contrast (Iopamidol 370) (0.5 - 2.0 mL/kg, 80mL maximum volume) will be injected with mechanical injector followed by 30ml saline flush.
- Scan parameters: slice collimation of 64 X 0.5 mm, a tube voltage of 120 kV, and a tube current of 70 mAs, gantry rotation time of 400 msec and slice thickness of 0.5 mm, Helical Retrospective gating.
- MDCT data analysis

    1. Coronary artery calcium score.
    2. Detection of the presence of Myocardial Bridge: as regard site, length, depth and degree of systolic obstruction.
    3. Coronary plaque assessment.

Results

The study was a single-center; randomized prospective, observational study we selected our patients among 1950 consecutive patients presented to the International Cardiac Center (ICC) scan between January 2015 and April 2016 referred to do 128-MDCT scan, all patients with Myocardial Bridge were selected till we collected 100 patients. With the exception of those with multiple ectopic beats, Heart rate greater than 75 beats per minute despite therapy, Severe lung disease, Renal failure, History of allergic reaction to contrast material, Atrial fibrillation, History of CABG or previous PCI.

Among the 100 patients we found 30 patients having Myocardial Bridge and proximal coronary artery atherosclerotic plaques included in Group (A) (30%), 70 persons having Myocardial Bridge without CAD included in Group (B) (70%).

Patient Demographics and Risk Factors

In the present study, patients with atheroscelerotic plaque (group A) (n = 30), mean age was 58.20± 9.38 years ranged from 35 to 73 years, 53.3% were males, 53.3% patients were diabetic, 83.3% were hypertensive, 66.7% was Dyslipidemic, 33.3% patients were smokers;60% had family history of CAD while in Myocardial bridge without atherosclerotic lesion group (group B) (n = 70), mean age was 55.21 ± 9.74 ranged from 33 to 77 years., 34.3% were males, 28.6% were diabetics, 71.4% were hypertensive, 45.7% was Dyslipidemic , 18.6%were smokers , 60% had family history of CAD.

	MB+ Lesion (n=30)		MB (n=70)		Test of sig.	p
	No.	%	No.	%		
Sex						
Female	14	46.7	46	65.7	c^2=3.175	0.075
Male	16	53.3	24	34.3		
Age						
Min. – Max.	35.0 – 73.0		33.0 – 77.0		t=1.421	0.159
Mean ± SD.	58.20 ± 9.38		55.21 ± 9.74			
Median	61.0		57.50			

Table 1: *Comparison between the two groups according to demographic data.*

c^2, p: c^2 and p values for Chi square test for comparing between the two groups

t, p: t and p values for Student t-test for comparing between the two groups

**: Statistically significant at p ≤ 0.05*

	MB+ Lesion (n = 30)		MB (n = 70)		χ^2	P
	No.	%	No.	%		
DM	16	53.3	20	28.6	5.589*	0.018*
HTN	25	83.3	50	71.4	1.587	0.208
Dyslipidemia	20	66.7	32	45.7	3.694	0.055
Smoking	10	33.3	13	18.6	2.584	0.108
Family history	18	60	42	60	0.000	1.000

Table 2: *Comparison between the two groups according to risk factors.*

c^2, p: c^2 and p values for Chi square test for comparing between the two groups

**: Statistically significant at p ≤ 0.0*

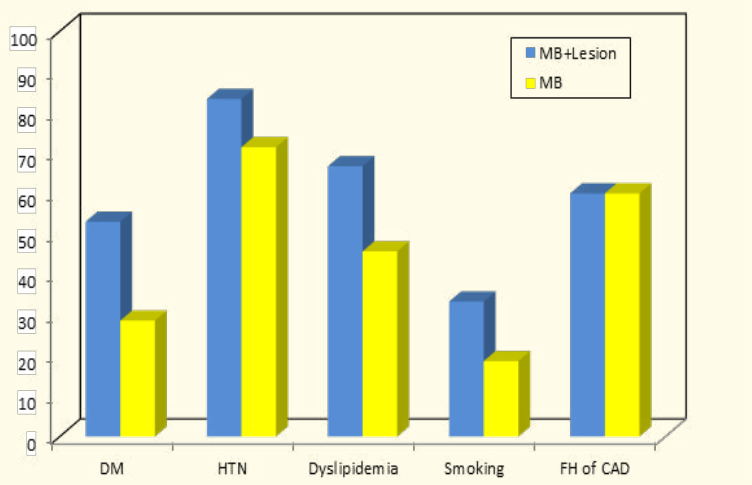

Figure 8: *Comparison between the two groups according to risk factors.*

MDCT Study

In present study, we assessed the myocardial bridge as regards the calcium score, site, length, depth and degree of systolic obstruction. we found that the mean calcium score in group (A) is 66.03 ± 81.33 and in group (B) is 4.76 ± 17.37. (P < 0.001). The mean length of MB in group (A) is 24.80 ± 11.93 mm and in group (B) is 24.21 ± 12.08 mm. (P = 0.679).

The mean depth of MB in group (A) is 3.81 ± 2.30 ranged from 1 to 11 mm and in group (B) is 2.91 ± 1.60 ranged from 1 to 8 mm. (P = 0.019). The mean Degree of systolic obstruction of MB in group (A) is 68.87± 19.42 ranged from 20% to 90%, and in group (B) is 31.07 ± 17.91 ranged from 0% to 90%. (P < 0.001).

Regarding the site in group A, MB was in Mid LAD in 20(66.7%) patients, Distal LAD in 10(33.3%) patients. In group B, MB was in Mid LAD in 36(51.4%) patients, Distal LAD in 25 (35.7%) patients and Proximal LAD in 8 (11.4%) patients, Mid-distal LAD 1(1.4%) patient. (^{MC}p = 0.150)

Calcium score	MB+ Lesion (n = 30)	MB (n = 70)	Z	p
Min. – Max.	0.0 – 428.0	0.0 – 122.0	6.334*	<0.001*
Mean ± SD.	66.07 ± 81.33	4.76 ± 17.37		
Median	67.0	0.0		

Table 3: *Comparison between the two groups according to calcium score.*
Z, p: Z and p values for Mann Whitney test for comparing between the two groups

**: Statistically significant at p ≤ 0.0*

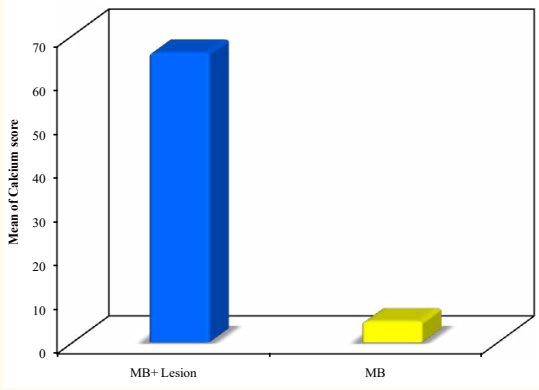

Figure 9: *Comparison between the two groups according to calcium score.*

	MB+ Lesion (n=30)		MB (n=70)		Test of sig.	p
	No.	%	No.	%		
Site						
Mid	20	66.7	37	52.9	χ^2=4.165	0.125
Distal	10	33.3	25	35.7		
Prox	0	0.0	8	11.4		
Length (mm)						

Min. – Max.	4.0 – 55.0	3.0 – 68.50	Z=0.414	0.679
Mean ± SD.	24.80 ± 11.93	24.21 ± 12.08		
Median	23.95	23.40		

Table 4: *Comparison between the two groups according to site and length of myocardial bridge.*

c^2, p: c^2 and p values for Chi square test for comparing between the two groups

Z, p: Z and p values for Mann Whitney test for comparing between the two groups

**: Statistically significant at p ≤ 0.0*

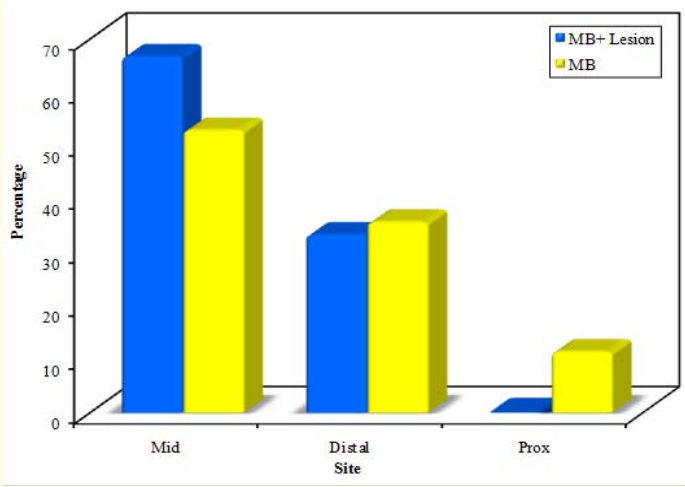

Figure 10: *Comparison between the two groups according to Site.*

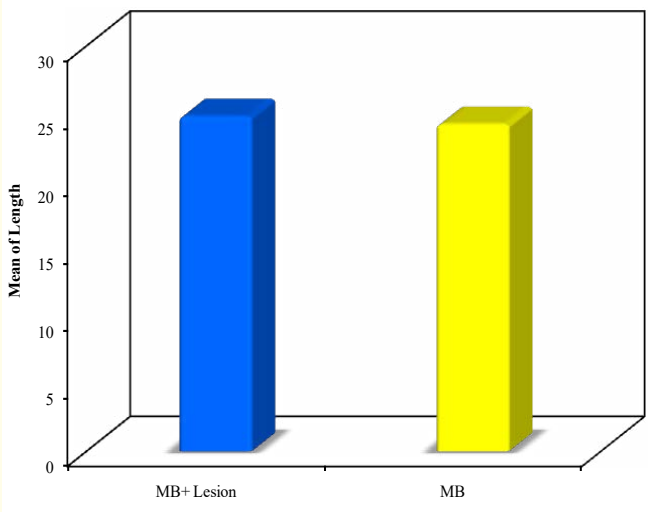

Figure 11: *Comparison between the two groups according to Length.*

	MB+ Lesion (n=30)	MB (n=70)	Z	p
Depth				
Min. – Max.	1.0 – 11.0	1.0 – 8.0	2.336*	0.019*
Mean ± SD	3.81 ± 2.30	2.91 ± 1.60		
Median	3.20	2.45		
Degree of systolic obstruction				
Min. – Max.	20.0 – 90.0	0.0 – 90.0	5.891*	<0.001*
Mean ± SD	60.87 ± 19.42	31.07 ± 17.91		
Median	60.0	30.0		

Table 5: *Comparison between the two groups according to depth and degree of systolic obstruction.*

Z, p: Z and p values for Mann Whitney test for comparing between the two groups

**: Statistically significant at p ≤ 0.05*

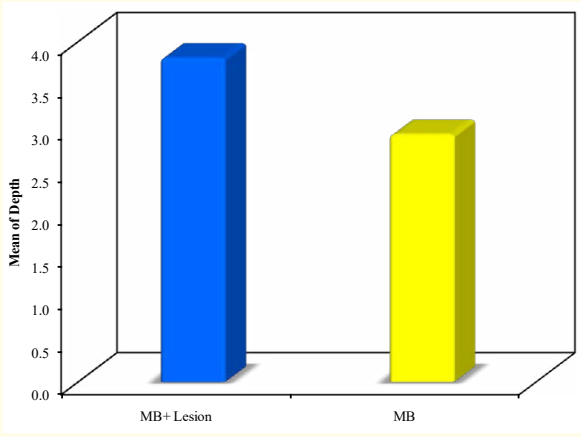

Figure 12: *Comparison between the two groups according to depth.*

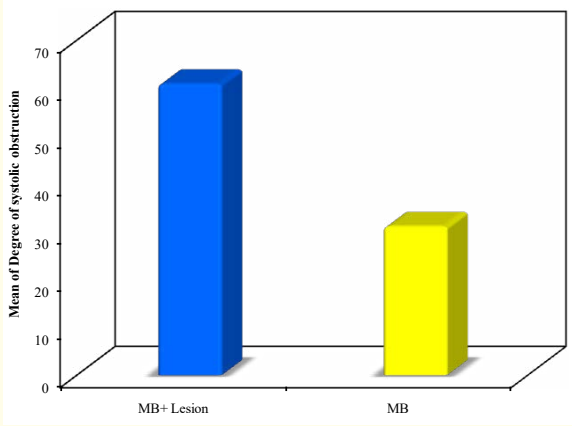

Figure 13: *Comparison between the two groups according to degree of systolic obstruction.*

The anatomical distribution of the affected Coronary Artery

The atherosclerotic plaque in group A was in LAD in 30(100%) of patients, in LM in 2(6.7%) of patients, in RCA in 3(10%) of patients, in Diagonal in 1(3.3%) of patients and in LCX in 1(3.3%) of patients.

Coronary plaque	No.	%
LAD	30	100
LM	2	6.7
RCA	3	10.0
Diagonal	1	3.3
LCX	1	3.3

Table 6: *Distribution of the studied cases according to coronary plaque in groupA.*

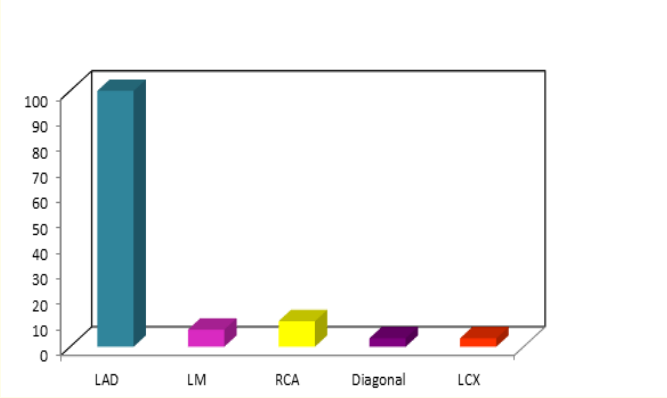

Figure 14: *Distribution of the studied cases according to coronary plaque in MB + Lesion group (n = 30).*

The atherosclerotic plaque was in Mid LAD in 5(16.7%) of patients, in Proximal LAD in 28(93.3%) of patients and in Distal LAD in 2(6.7%) of patients.

We found that the plaque site in LAD in group A was most common in Proximal LAD.

Segment	No.	%
Mid	5	16.7
Prox.	28	93.3
Distal	2	6.7

Table 7: *Distribution of the studied cases according to segment in MB+ Lesion group (n=30).*

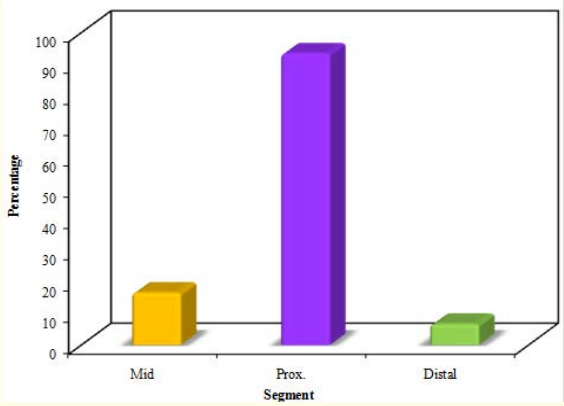

Figure 15: *Distribution of the studied cases according to segment in group A.*

The plaque characteristics

In group A the percent of obstruction mean was 42.66 ± 24.69% ranging from 14% to 100% with median 33%, Total Plaque Volume (TPV) was 186.26 ± 101.13 ranging from 65 to 505 with median 150, the vessel volume was 317.07 ± 168.96 ranging from 116.70 to 806 with median 104.

The plaque burden was 56.14 ± 11.80% ranging from 23% to 77.40% with median 59%.

	Min. – Max.	Mean ± SD.	Median
Obstruction%	14.0 – 100.0	42.66 ± 24.69	33.0
TPV	65.0 – 505.0	186.26 ± 101.13	150.0
Vessel volume	116.70 – 806.0	317.07 ± 168.96	247.0
Lumen volume	40.0 – 301.0	120.53 ± 64.59	104.0
Plaque Burden%	23.0 – 77.40	56.14 ± 11.80	59.0

Table 8*: Descriptive analysis of the studied cases according to different parameters in groupA.*

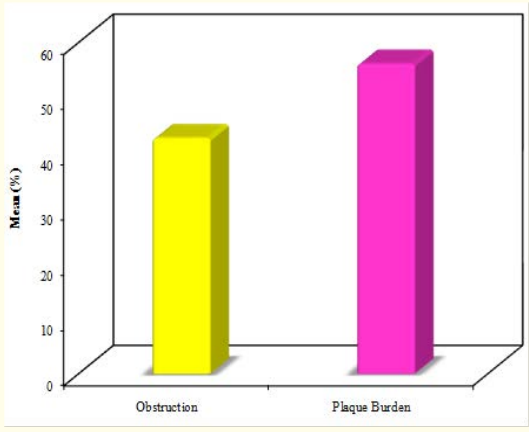

Figure 16: *Descriptive analysis of the studied cases according to Obstruction% and Plaque Burden% in group A.*

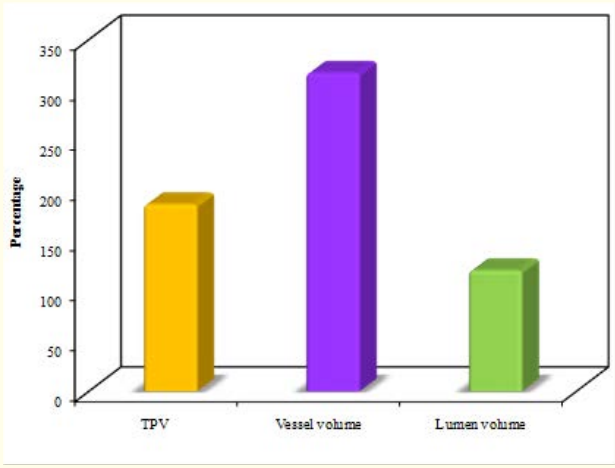

Figure 17: *Descriptive analysis of the studied cases according to Vessel volume and Lumen volume in (group A).*

The atherosclerotic plaque type was LDPV in 10(33.3%) of patients, MDPV in 21(70%) of patients and HDPV in 4(13.3%) of patients.

	No.	%
LDPV	10	33.3
MDPV	21	70.0
HDPV	4	13.3

Table 9: *Distribution of the studied cases according to obstruction in MB + Lesion group (n = 30).*

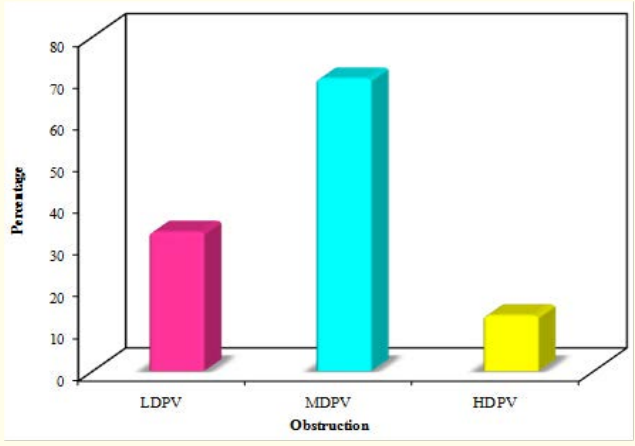

Figure 18: *Distribution of the studied cases according to obstruction in group A.*

	Simple Regression Analysis				Multiple Regression Analysis				
	OR	95% CI		p	B	OR	95% CI		p
		LL	UL				LL	UL	
Age	1.034	0.987	1.084	0.159	-0.082	0.921	0.807	1.052	0.224
Sex	0.457	0.191	1.090	0.078	-0.962	0.382	0.034	4.257	0.434
DM	2.857	1.179	6.923	0.020*	3.607	36.866	2.071	656.162	0.014*
HTN	2.00	0.672	5.956	0.213	-	-	-	-	-
Dyslipidemia	2.375	0.972	5.801	0.058	-0.948	0.387	0.057	2.618	0.331
Smoking	2.192	0.832	5.778	0.112	0.369	1.446	0.100	20.947	0.787
Family history	1.000	0.418	2.394	1.000	-	-	-	-	-
Calcium score	1.047	1.027	1.067	<0.001*	0.057	1.059*	1.027	1.091	<0.001*
Length	1.004	0.969	1.041	0.821	-	-	-	-	-
Depth	1.283	1.018	1.615	0.035*	-0.575	0.563	0.309	1.024	0.060
Degree systolic obstruction	1.075	1.045	1.107	<0.001*	0.160	1.173*	1.070	1.286	0.001*

Table 10: *Multivariate analysis logistic regression for atherosclerotic Lesion.*

B: Unstandardized Coefficients

SE: Standard Error

OR: Odds ratio

CI: Confidence interval

LL: Lower limit

UL: Upper Limit

**: Statistically significant at p ≤ 0.05*

	Obstruction%	
	r_s	p
Length	-0.038	0.827
Depth	0.351*	0.039*
Degree of systolic obstruction	0.111	0.526

Table 11: *Correlation between obstruction% and different parameters in MB (n = 35).*

rs: Spearman coefficient

**: Statistically significant at p ≤ 0.05*

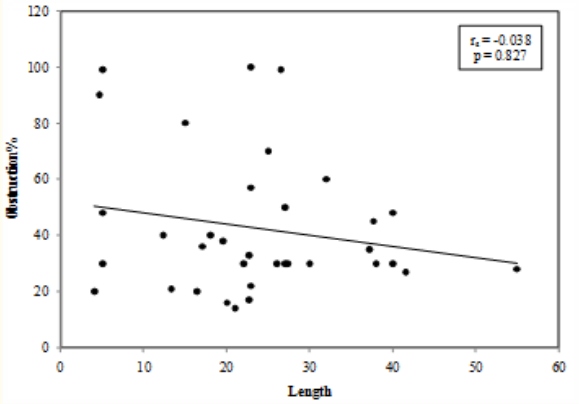

Figure 19: *Correlation between obstruction% and Length in MB (n = 35).*

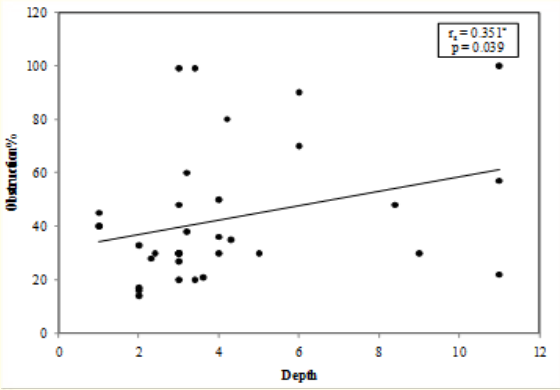

Figure 20: *Correlation between obstruction% and Depth in MB (n = 35).*

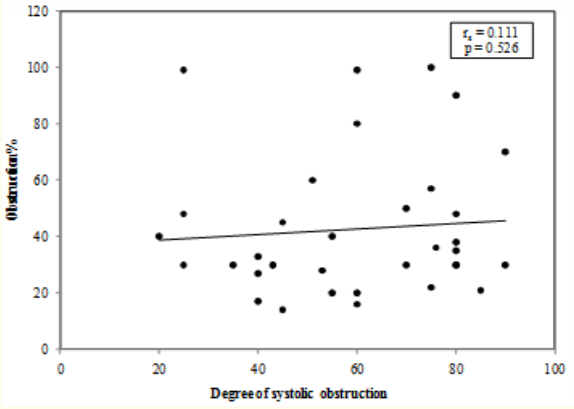

Figure 21: *Correlation between obstruction% and Degree of systolic obstruction in MB (n = 35).*

Discussion

Myocardial bridging is a common anatomic variant on coronary CTA. The frequency of bridging on coronary CTA is higher than that reported for ICA, because of the strong reliance of the latter of the finding of systolic compression of the involved segment in establishing the diagnosis. The lower prevalence of myocardial bridges identified on invasive angiography compared to MSCT may be related to the fact that the pathognomonic "milking-phenomenon" is an indirect diagnostic criterion because invasive angiography does not allow direct visualization of the myocardium.

Emerging data suggest that certain anatomic characteristics of myocardial bridges, such as length and depth, may contribute to the development of atherosclerosis and be related to subsequent cardiac events as well to the presence of ischemia.

Location and morphology (length, thickness, and diameter) by using 128-MDCT.

Myocardial bridges vary in size with a reported length ranging from 8 - 50 mm by coronary CTA, and a depth between and 1 – 4 mm by coronary CTA. Longer and deeper bridges and those that exhibit greater degrees of systolic compression (> 70%) are more common in symptomatic patients [40].

Mechanisms for Atherosclerosis in the Segment Proximal to the Bridge

Hemodynamic forces may explain atherosclerotic plaque formation at the entrance to the tunneled segment. There, the endothelium is flat, polygonal, and polymorph, indicating low shear, whereas in the tunneled segment, the endothelium has a helical, spindle-shaped orientation along the course of the segment as a sign of laminar flow and high shear [41].

Low shear stress may induce the release of endothelial vasoactive agents such as endothelial nitric oxide synthase (eNOS), endothelin-1 (ET-1), and angiotensin-converting enzyme (ACE) [42].

Their levels were significantly higher in proximal and distal segments compared with the tunneled segment. Thus, low shear stress may contribute to atherosclerotic plaque formation proximal to the bridge, whereas high shear stress may have a protective role within the tunneled segment [43-45].

In addition, an increase in local wall tension and stretch may induce endothelial injury and plaque fissuring with subsequent thrombus formation in the proximal segment which is supported by autopsy and clinical observations.

Conclusion

- Myocardial bridge was usually located over the Mid segment of the left anterior descending coronary artery
- Certain anatomic characteristics of myocardial bridges, such as depth and degree of systolic obstruction, may contribute to the development of atherosclerosis.

Bibliography

1. Angelini P., *et al*. "Coronary anomalies: incidence, pathophysiology, clinical relevance". *Circulation* 105 (2002): 2449-2454.
2. Angelini P., *et al*. "Myocardial bridges: a review". *Progress in Cardiovascular Diseases* 26.1 (1983): 75-88.
3. Reyman HC. "Diss. de vasis cordis propriis". *Bibliotheca Anatomica* 2 (1737): 359-379.
4. Portmann WC and Iwig J. "Die intramurale koronarie im angiogram". *Fortschr Röntgenstr* 92 (1960):129–132.
5. Geirnger E. "The mural coronary". *American Heart Journal* 41.3 (1951): 359-368.

6. Polacek P and Kralove H. "Relation of myocardial bridges and loops on the coronary arteries to coronary occulsions". *American Heart Journal* 61.1 (1961): 44-52.

7. Gittenberger-de Groot AC., *et al*. "Epicardium-derived cells contribute a novel population to the myocardial wall and the atrioventricular cushions". *Circulation Research* 82.10 (1998): 1043-1052.

8. Henry Gray. "Gray's Anatomy 38th ed". *Edinberg: Churchill Livingstone* (1999):1505-1510.

9. Reig J and Petit M. "Main Trunk of the Left Coronary Artery: Anatomic Study of the Parameters of Clinical Interest". *Clinical Anatomy* 17.1 (2004): 6-13.

10. Hort W. "Anatomie und Pathologie der Koronararterien. B. Muskelbrücken der Koronararterien". In: W. Hort, Hrsg. Pathologie desEndokards, der Koronararterien und des Myokards. *Berlin, Germany: Springer-Verlag, Heidelberg* (2000): 220-231.

11. Van Nie CJ and Vincent JG. "Myocardial bridges in animals". *Anatomia, Histologia, Embryologia* 18.1 (1989): 45-51.

12. Fishman MC and Chien KR. "Fashioning the vertebrate heart: earliest embryonic decisions". *Development* 124.11 (1997): 2099-2117.

13. Dettman RW., *et al*. "Common epicardial origin of coronary vascular smooth muscle, perivascular fibroblasts, and intermyocardial fibroblasts in the avian heart". *Developmental Biology* 193.2 (1998): 169-181.

14. La Grutta L., *et al*. "Prevalence of myocardial bridging and correlation with coronary atherosclerosis studied with 64-slice CT coronary angiography". *Radiologia Medica* 114.7 (2009): 1024-1036.

15. Ko SM., *et al*. "Incidence and clinical significance of myocardial bridging with ECG-gated 16-row MDCT coronary angiography". *International Journal of Cardiovascular Imaging* 24.4 (2008): 445-452.

16. Soran O., *et al*. "The incidence and significance of myocardial bridge in a prospectively defined population of patients undergoing coronary angiography for chest pain". *Tokai Journal of Experimental and Clinical Medicine* 25.2 (2000): 57-60.

17. Ishimori T., *et al*. "Myocardial bridges in man: clinical correlations and angiographic accentuation with nitroglycerin". *Catheterization and Cardiovascular Diagnosis* 3.1 (1977): 59-65.

18. Hongo Y., *et al*. "Augmentation of vessel squeezing at coronary-myocardial bridge by nitroglycerin: study by quantitative coronary angiography and intravascular ultrasound". *American Heart Journal* 138.2 (1999): 345-350.

19. Ishii T., *et al*. "The significance of myocardial bridge upon atherosclerosis in the left anterior descending coronary artery". *Journal of Pathology* 148.4 (1986): 279-291.

20. Kawawa Y., *et al*. "Detection of myocardial bridge and evaluation of its anatomical properties by coronary multislice spiral computed tomography". *European Journal of Radiology* 61.1 (2007): 130-138.

21. Lubarsky L., *et al*. "Evaluation of myocardial bridging of the left anterior descending coronary artery by 64-slice multidetector computed tomographic angiography". *American Journal of Cardiology* 100.7 (2007): 1081-1082.

22. Wang MH., *et al*. "Myocardial bridging detection by non-invasive multislice spiral computed tomography: comparison with intravascular ultrasound". *Chinese Medical Journal* 121.1 (2008): 17-21.

23. Kramer JR., *et al*. "Clinical significance of isolated coronary bridges: benign and frequent condition involving the left anterior descending artery". *American Heart Journal* 103.2 (1982): 283-288.

24. Souibri K and Grollier G. "Infarction due to myocardial bridging". *New England Journal of Medicine* 353 (2005): 1147.

25. David M and Wolf JE. "Acute myocardial infarction related to myocardial bridging". *European Heart Journal* 16.12 (1995): 2002-2003.

26. Takamura K., *et al*. "Anatomical characteristics of myocardial bridge in patients with myocardial infarction by multi-detector computed tomography". *Circulation Journal* 75.3 (2011): 642-648.

27. Faruqui AM., *et al*. "Symptomatic myocardial bridging of coronary artery". *American Journal of Cardiology* 41.7 (1978): 1305-1310.

28. Gow RM. "Myocardial bridging: does it cause sudden death?" *Cardiac Electrophysiology Review* 6.1-2 (2002): 112-114.

29. Desseigne P., *et al*. "Myocardial bridging on the left anterior Descending coronary artery and sudden death. Apropos of 19 cases with autopsy [in French]". *Archives Des Maladies Du Coeur Et Des Vaisseaux* 84.4 (1991): 511-516.

30. Morales AR., *et al*. "Intramural LAD: significance of depth of the muscular tunnel". *Human Pathology* 24.7 (1993): 693-701.

31. Erbel R., *et al.* "Coronary artery shape and flow changes induced by myocardial bridging". *Assessment by Intravascular Ultrasound Echocardiography* 10.1 (1993): 71-77.

32. Yetman AT., *et al*. "Myocardial bridging in children with hypertrophic cardiomyopathy—a risk factor for sudden death". *New England Journal of Medicine* 339.17 (1998): 1201-1209.

33. Voelker W., *et al*. "Myocardial bridges at multiple sites over the left coronary artery in a patient with hypertrophic cardiomyopathy". *International Journal of Cardiology* 23.2 (1989): 258-260.

34. Achrafi H. "Hypertrophic cardiomyopathy and myocardial bridging". *International Journal of Cardiology* 37.1 (1992): 111-112.

35. Schwarz ER., *et al*. "Functional, angiographic and intracoronary Doppler flow characteristics in symptomatic patients with myocardial bridging: effect of short-term intravenous beta-blocker medication". *Journal of the American College of Cardiology* 27.7 (1996): 1637-1645.

36. Doshi AA., *et al*. "Drug eluting stent implantation for the treatment of symptomatic myocardial bridging is associated with favorable peri-procedural results and short-term outcomes". *International Journal of Cardiology* 118.3 (2007): e87-e88.

37. Hillman ND., *et al*. "Supraarterial decompression myotomy for myocardial bridging in a child". *Annals of Thoracic Surgery* 68.1 (1999): 244-246.

38. Kim PJ., *et al.* "Frequency of myocardial bridges and dynamic compression of epicardial coronary arteries: a comparison between computed tomography and invasive coronary angiography". *Circulation* 119.10 (2009): 1408-1416.

39. Flynn MS., *et al*. "Intramyocardial muscle bridging of the coronary artery - an examination of a diastolic "spike and dome" pattern of coronary flow velocity". *Catheterization and Cardiovascular Diagnosis* 32.1 (1994): 36 -39.

40. Mookadam F., *et al*. "Clinical relevance of myocardial bridging severity: single center experience". European *Journal of Clinical Investigation* 39.2 (2009): 110-115.

41. Ishii T., *et al*. "The effects of a myocardial bridge on coronary atherosclerosis and ischemia". *Journal of Pathology* 185.1 (1998): 4-9.

42. Masuda T., *et al.* "The effect of myocardial bridging of the coronary artery on vasoactive agents and atherosclerosis localization". *Journal of Pathology* 193.3 (2001): 408-414.

43. Malek AM., *et al*. "Hemodynamic shear stress and its role in atherosclerosis". *Journal of the American Medical Association* 282.21 (1999): 2035-2042.

44. Ge J., *et al*. "High wall shear stress proximal to myocardial bridging and atherosclerosis: intracoronary ultrasound and pressure measurements". *British Heart Journal* 73.5 (1995): 462-465.

45. Klues HG., *et al*. "Disturbed intracoronary hemodynamics in myocardial bridging. Early normalization by intracoronary stent placement". *Circulation* 96.9 (1997): 2905-2913.

Volume 2 Issue 6 November 2016