

Proportion of Myocardial Bridge in a Tertiary Care Hospital of North India: A Retrospective Observational Study

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ABSTRACT

Introduction: Myocardial bridging, a congenital coronary anomaly, is a condition in which a segment of a major epicardial coronary artery runs intramurally through the myocardium. During systole, the coronary artery is compressed and can cause angina, arrhythmia, depressed left ventricular function, myocardial stunning and sudden death.

Aim: To find the proportion of Myocardial Bridge (MB) in patients admitted for cardiac evaluation in a tertiary care hospital of north India.

Materials and Methods: A retrospective, observational study was performed in the Cardiac Care Unit of the Adesh institute of Medical Sciences and Research Bathinda from January 2017 to December 2020. A total of 3800 adult patients of both sexes, who underwent diagnostic coronary angiography were evaluated for MB between January 2017 to December 2020. The data was collected from the patients and statistically analysed on 15th April 2021.

Results: The proportion of MB was 208 (5.50%), in which 74.51% were males and 25.49% were females. Dominance-wise, 72.11% were right dominant, 22.60% were left dominant and 5.29% had balanced circulation. The incidence of myocardial bridging was 3.95% in right dominant, 1.24% in left dominant and 0.28% in balanced dominant patients. Total 199 (95.67%) MB were located on the Left Anterior Descending Artery (LAD) of which mid LAD were in 186 (89.42%), distal LAD in 9 (4.45%), proximal LAD in 4 (1.95%) and on diagonal (D1) it was 9 (4.45%). Double bridge was observed in 6 (2.89%) cases in proximal and distal regions of LAD.

Conclusion: Clinical suspicion of a MB should be considered in young patients with typical or atypical chest pain, where the probability of atherosclerosis is low and who there are no other cardiovascular risk factors. There was higher incidence of MB in proximal distal segments of LAD and diagonal arteries.

Keywords: Angiogram, Congenital coronary anomalies, Left anterior descending artery

INTRODUCTION

Coronary arteries are normally epicardial in position. In some patients a segment of coronary artery is crossed by few cardiac muscle fibers. This pattern of arrangement of cardiac muscle fibers over a segment of coronary artery is known as Myocardial Bridge (MB), which is a congenital anomaly. The segment of coronary artery that has intramural course is known as tunneled artery or intramural. This was first seen in autopsy by Reyman HC in 1737 [1]. First MB was reported by Geiringer E in 1951 during anatomical dissection [2]. The systolic compression of epicardial coronary artery by the MB was first seen on coronary angiography by Portmann WC and Iwig I in 1960 [3]. The incidence of MB on angiographic studies range from 0.5-8.5% [4-6]. During autopsy the incidence ranges from 5.5-90% [7-13]. Many authors stated that MB may be protective on the coronary artery at the site of bridge [14,15]. Although MB is asymptomatic in many patients, its association with myocardial ischaemia, stunting, heart failure, myocardial infarction and sudden cardiac death had been reported [16,17].

The MB are generally located on the middle segment of Left Anterior Descending artery (LAD) involving different lengths of the coronary arteries and varying depths of myocardial wall [18]. The coronary angiography shows systolic compression of epicardial coronary artery at site of MB and leads to coronary obstruction. The typical angiographic finding in myocardial bridging is systolic narrowing of an epicardial artery [18].

The percentage of coronary obstruction depends on various factors such as length of MB, thickness of MB and degree of cardiac contractility [19,20]. Previously thought to be benign and asymptomatic condition, but recently found to be associated with angina, myocardial ischaemia, acute coronary syndrome, left ventricular dysfunction/ stunning, supraventricular and ventricular arrhythmias, ventricular septal rupture and sudden cardiac death [21-26]. The present study

was carried out to find out the proportion of MB in patients admitted for cardiac evaluation in a tertiary care centre of North India.

MATERIALS AND METHODS

After approval of Institutional Ethics Committee wide letter number-(AU/EC/FM/2021/134), this retrospective, observational study was conducted in the Cardiac Care Unit, of the Adesh institute of Medical Sciences and Research Bathinda from January 2017 to December 2020. As per hospital protocol these patients were taken up for intervention, while the selection criteria for the study depended upon the data that was available (being retrospective). The data was analysed on 15th April 2021.

Inclusion and Exclusion criteria: A total of 3800 adult patients of both sexes who underwent diagnostic conventional coronary angiography were included in the study. Patients with total occlusion bypass surgery and artifacts were excluded from the study.

Coronary artery disease and presence of MB were recorded. As per hospital protocol, MB was identified based on narrowing of coronary artery in systolic phase resulting in at least 50% reduction of luminal diameter in comparison with the diastolic phase. The diameter of vessel was measured during in end-systolic and enddiastolic phases with an electronic caliper after magnification. All the measurements were recorded in the left anterior oblique position.

STATISTICAL ANALYSIS

The relevant data was collected from the available record of patients. The data was qualitatively analysed and expressed as frequency distribution of percentages.

RESULTS

Total of 3800 patients, who went for coronary evaluation, were included in the study. Nineteen patients were excluded due to total occlusion (7), bypass surgery (8) or artifacts (4). Of all patients, 2712 (71.37%) were males and 1088 (28.63%) were females. Total 82.26% patients had normal coronary arteries, single vessel disease, 2.53% double vessel disease and 1.78% had triple vessel disease.

There were a total of 208 MBs (5.50%) of which 155 were males and 53 were females. Among all the MB patients, 199 (95.67%) were located LAD of which 186 (89.42%) were in mid LAD, 9 (4.45%) on distal LAD, 4 (1.95%) on proximal LAD and 9 (4.45%) were on diagonal (D1). In 6 (2.89%) patients, double bridges in proximal and distal parts of LAD was observed. Total 185 (88.95%) patients with MB had right, 20 (9.61%) left and 3 (1.44%) had balanced dominance [Table/Fig-1].

| Dominance | Right | n (%) | Left n (%) | | Balanced (%) | | Total | |
|---|-----------------|-----------------|---------------|--------------|--------------|-------------|-----------------|--|
| Sex | Male | Female | Male | Female | Male | Female | % | |
| Bridge | | | | | | | | |
| Present | 158 (2.89) | 27 (1.06) | 12 (0.93) | 8 (0.31) | 2 (0.26) | 1 (0.02) | 208 (5.50) | |
| Absent | 2295 (60.40) | 985 (25.93) | 241 (6.34) | 42 (1.10) | 21 (0.55) | 8 (0.21) | 3592 (94.50) | |
| Total | 2405 (63.29) | 1025 (26.99) | 276 (7.27) | 54 (1.41) | 31 (0.81) | 9 (0.23) | 3781 (100) | |
| Diagnosis | | | | | | | | |
| Normal | 2102 (55.31) | 800 (21.01) | 175 (4.50) | 40 (1.05) | 10 (0.26) | 9 (0.23) | 3126 (82.26) | |
| SVD | 260 (6.84) | 148 (3.89) | 76 (2.0) | 8 (0.2) | 10 (0.26) | 0 | 502 (13.21) | |
| DVD | 22 (0.57) | 50 (1.31) | 22 (0.56) | 4 (0.1) | 6 (0.15) | 0 | 104 (2.53) | |
| TVD | 31 (0.81) | 27 (0.71) | 3 (0.08) | 2 (0.05) | 5 (0.13) | 0 | 68 (1.78) | |
| Vessel | | | | | | | | |
| LAD | | | | | | | | |
| Middle | 148 (71.15) | 20 (9.61) | 9 (4.33) | 6 (2.88) | 2 (0.96) | 1 (0.48) | 186 (89.42) | |
| Distal | 4 (1.92) | 4 (1.92) | 1 (0.48) | 0 | 0 | 0 | 9 (4.33) | |
| Proximal | 2 (0.96) | 2 (0.96) | 0 | 0 | 0 | 0 | 4 (1.92) | |
| Diagonal | 4 (1.92) | 1 (0.48) | 2 (0.96) | 2 (0.96) | 0 | 0 | 9 (4.33) | |
| Surgical treatment | None | | | | | | | |
| Stenting | None | | | | | | | |
| [Table/Fig-1]: Patient characteristics and diagnosis. SVD: Single vessel disease; DVD: Double vessel disease; TVD: Triple vessel disease; LAD: Left anterior descending | | | | | | | | |

Twenty-two (10.57%) patients presented with chest pain, 25 (12.01%) with dyspnoea on exertion and 161 (77.42%) with atypical chest pain. The male patients with MB were of age between 36-55 years and female patient's age between 42-58 years [Table/Fig-2].

| Variables | Male | Female | Total N (%) | | | | |
|--|-------------|------------|-------------|--|--|--|--|
| Age (years) mean±SD | 44±10.6 | 46±10.1 | | | | | |
| Symptoms N (%) | | | | | | | |
| Chest pain | 15 (7.21) | 7 (3.36) | 22 (10.57) | | | | |
| Dyspnoea on exertion | 20 (9.61) | 5 (2.40) | 25 (12.01) | | | | |
| Atypical chest pain | 120 (57.70) | 41 (19.72) | 161 (77.42) | | | | |
| Total | 155 (74.52) | 53 (25.48) | 208 | | | | |
| Table (Fig. Ob. Age and sumptome of patients with Musescript Dridge (MD) | | | | | | | |

[Table/Fig-2]: Age and symptoms of patients with Myocardial Bridge (MI

DISCUSSION

The MB is a common finding during routine coronary angiogram. Few previous studies had revealed the cardio protective role of MB, while others showed no protective role of MB. The incidence of MB is found to be greater at autopsy studies than conventional angiographic evaluation [7-13]. Although this malformation is present at birth, symptoms usually develop after the third decade; the reason for which is not clear [15]. The thickness, length of bridge and arrangement of cardiac muscle fibers over the artery affects the appearance of MB on coronary angiography [17]. Use of quality cineangiographic equipment and best techniques with particular attention devoted to examining the phasic changes of coronary diameter might allow a higher number of MB to be identified [17].

The MBs were considered to be a benign condition, but recently these have been associated with serious clinical complications like ischaemia and acute coronary syndromes [19], coronary spasm [20], ventricular septal rupture [21], arrhythmias [22], exercise-induced atrioventricular conduction blocks [23], transient ventricular dysfunction [24] and sudden death [25]. The prognosis of patients with MB, therefore, is not as benign as it was believed to be in the past. In the present study, most of the patients presented with atypical chest pain (77.42%) followed by dyspnoea on exertion (12.01%) and chest pain (10.57%).

Myocardial bridging is generally confined to the mid segment of LAD artery [26]; it is less frequently located in the circumflex artery and is occasionally seen in the right coronary artery. The most commonly involved vessel is left anterior descending artery (middle segment) followed by the left circumflex and the right coronary artery [18,26,27]. The present study found maximum number of MB in mid LAD followed by distal and proximal and diagonal region. Nine patients had MB in mid diagonal (D1). Six patients had double bridges in proximal and distal LAD. The patients with MB were of relatively younger age group. Mid-LAD was predominantly the site for MB in the present study which is similar to the study by Mavi A et al., but in contrast to Cay S et al., where an almost equal distribution was observed in either segments [27,28].

Therapeutic approaches that have been attempted for myocardial bridging include beta-blockers, calcium channel blockers, stents [29-36], minimally invasive Coronary Artery Bypass Grafting (CABG) [33], and surgical myotomy [34-39].

Some authors [31,39-41] reported the incidence of MB to be ranging from 0.6%-3.17%. In the present study, the incidence of MB was 5.50% that is higher than that reported in the previous studies in Indian scenario. The MB constricts the coronary arteries in systole whereas blood flow is normally in diastole; the mechanism of production of ischaemic heart disease has been debated. Bourassa MG et al., with the help of new cardiologic investigation facilities, found that systolic coronary artery compression leads to mid to late reduction of their diastolic diameter which might be responsible for cardiac compromise [42]. The segment proximal to the bridge frequently shows atherosclerotic changes, although the tunneled segment is typically spared. This is supported by effects at cellular and ultrastructural level [43,44].

The MB is a physiological entity offering maximum resistance to the coronary blood flow. In a normal condition, this resistance is negated by vessel dilatation in response to local metabolites. But with increasing age, this phenomenon is compromised which may lead to myocardial ischaemia. Hence, the knowledge of MBs is essential for cardiologists to detect aetiology of different heart related problems, to plan the mode of treatment and to predict their prognosis. Surgical approaches like coronary bypass, unroofing and myotomy are the treatment of choice for symptomatic MB [45]. High inflation pressures may be required to optimally implant the stent, which increases the risk of coronary perforation. The data collected until now demonstrated that the treatment of MB with stent implantation is not free from potential complications [46,47]. In the index study, none of the patients required percutaneous coronary or surgical intervention.

Limitation(s)

As this was a retrospective study, the patients with MB could not be followed-up for progression of disease.

CONCLUSION(S)

Clinical suspicion of MB should be considered in young patients with typical or atypical chest pain, where the probability of atherosclerosis is low and there are no other cardiovascular risk factors. The MB is a congenital anomaly but is symptomatic in third decade of life. Previously thought to be a benign condition, but it can be symptomatic. The recognition of this condition is very important and so is its management. This study observed higher proportion of MB in proximal and distal segment of LAD and diagonal arteries.

REFERENCES

- Reyman HC. Disertatio de vasis cordis propriis. Med Diss Univ Göttingen. 7th Sept 1737; 1-32.
- [2] Geiringer E. The mural coronary. Am Heart J. 1951;41:359-68.
- [3] Portmann WC lwig J. Intramural coronary vessels in the angiogram. Fortschr Rontgenstr. 1960;92:129-33.
- [4] JuilliereY, Berder V, Suty-Selton C, Buffet P, Danchin N, Cherrier F. Isolated myocardial bridges with angiographic milking of the left anterior descending coronary artery: A long term follow up study. Am Heart J. 1995:129(4):663-65.
- [5] Rossi L, Danber B, Nadasio GP, Arbustini F, Paris B, Vassanelli C, et al. Myocardial bridges and ischaemic heart disease. Eur Heart J. 1980;1(4):239-45.
- [6] Kramer JR, Kitazume H, Proudfit WL, Sones FM. Clinical significance of isolated coronary bridges and frequent condition involving the left anterior descending artery. Am Heart J. 1982;103(2):283-88.
- [7] Rajendra Prasad VK, John JT, Supriya NK. Occurrence and anatomical distribution of myocardial bridges and co-relation with sudden death and coronary atherosclerosis. International journal of Recent Trends in science and Technology. 2013;8(3):238-39.
- [8] Loukas M, Curry B, Bowers M, Louis RG, Bartczak A, Kiedrowski M, et al. The relationship of myocardial bridges to the coronary artery dominance in the adult human hearts. J Anat. 2006;209(1):43-50.
- [9] Saidi H, Ongeti WK, Ogengo J. Morphology of human myocardial bridges and coronary artery disease. Afr Health Sci. 2010;10(3):242-47.
- [10] Acunã LE, Aristeguieta LM, Tellez SB. Myocardial description and clinical implication of myocardial bridges: An anatomical study in Colombians. Arq Bras Cardiol. 2009;92(4):242-48.
- [11] Bandyopadhyay M, Das P, Baral K, Chakroborty P. Morphological study of myocardial bridges on the coronary arteries. Indian Journal of Thoracic and Cardiovascular Surgery. 2010;26(3):193-97.
- [12] Kosinski A, Grzybiak M. Myocardial bridges in the human heart: Morphological aspect. Folia Morphol. 2001;60(1):65-68.
- [13] Chandra Sekhar KT, Harsha BR. A cadaveric study on muscle bridges in human hearts. IJAPB. 2015;2(3):01-06.
- [14] Ishii T, Hosoda Y. The significance of myocardial bridge upon atherosclerosis in the left anterior descending coronary artery. J Path. 1986;148:279-91.
- [15] Laifer LI, Weiner BH. Percutaneous trans luminal coronary angioplasty of a coronary artery stenosis at the site of myocardial bridging. Cardiology. 1991;79:245-48.
- [16] Kuhn FE, Rgan K, Mohler ER, Ill. Satler LF, Lu Dy, Rackley CE. Evidence for endothelial dysfunction and enhanced vasoconstrution in myocardial bridges. Am Heart J. 1991;122:1764-66.
- [17] Baptista CA, Didio LJ. The relationship between the directions of myocardial bridges and of the branches of the coronary arteries in the human heart. Surg Radiol Anat. 1922;14:137-40.
- [18] Arjomand H, Alsalman J, Azian J, Admin D. Myocardial bridging of left circumflex coronary artery associated with acute myocardial infarction. J Invasive Cardiol. 2000;12:431-34.
- [19] Mazzu A, Di Tano G, Cogode R, Lo Presti G. Myocardial bridging involving more than one site of the left anterior descending coronary artery: An uncommon cause of acute ischaemic syndrome. Cathet Cardiovasc Diagn. 1995;34:329-32.
- [20] Berry JF, von Mering GO, Schmalfuss C, Hill JA, Kerensky RA. Systolic compression of the left anterior descending coronary artery: A case series, review of the literature, and therapeutic options including stenting. Catheter Cardiovasc Interv. 2002;56:58-63.
- [21] Tio RA, Ebels T. Ventricular septal rupture caused by myocardial bridging. Ann Thorac Surg. 2001;72:1369-70.

- [22] Feld H, Guadanino V, Hollander G, Greengart A, Lichstein E, Shani J. Exerciseinduced ventricular tachycardia in association with a myocardial bridge. Chest. 1991;99:1295-96.
- [23] den Dulk K, Brugada P, Braat S, Heddle B, Wellens HJ. Myocardial bridging as a cause of paroxysmal atrioventricular block. J Am Coll Cardiol. 1983;1:965-69.
- [24] Galli M, Politi A, Zerboni S. "Functional myocardial bridging" and "hyperkinetic state": A rare association as a cause of acute myocardial infarction" G Ital Cardiol. 1997;27:1286-89.
- [25] Cutler D, Wallace JM. Myocardial bridging in a young patient with sudden death. Clin Cardiol. 1997;20:581-83.
- [26] Faruqui AM, Maloy WC, Felner JM, Schlant RC, Logan WD, Symbas P. Symptomatic myocardial bridging of coronary artery. Am J Cardiol. 1978;41:1305-10.
- [27] Mavi A, Sercelik A, Ayalp R, Karben Z, Batyraliev T, Gumusburun E. The angiographic aspects of myocardial bridges in Turkish patients who have undergone coronary angiogram. Ann Acad Med Singapore. 2008;37:49-53.
- [28] Cay S, Oztürk S, Cihan G, Kisacik HL, Korkmaz S. Angiographic prevalence of myocardial bridging. Anadolu Kardiyol Derg. 2006;6:09-12.
- [29] Garg S, Brodison A, Chauhan A. Occlusive systolic bridging of circumflex artery. Catheter Cardiovasc Interv. 2000;51:477-78.
- [30] Zen K, Ito K, Tanabe T, Hikosaka T, Adachi Y, Kato S. Stent implantation in a case of myocardial bridging with resistant angina pectoris. Nihon Naika Gakkai Zasshi. 2001;90(5):874-76.
- [31] Harikrishnan S, Sunder KR, Tharakan J, Titus T, Bhat A, Sivasankaran S, et al. Clinical and angiographic profile and follow-up of myocardial bridges: A study of 21 cases. Indian Heart J. 1999;51(5):503-07.
- [32] Haager PK, Schwarz ER, vom Dahl J, Klues HG, Reffelmann T, Hanrath P. Long term angiographic and clinical follow up in patients with stent implantation for symptomatic myocardial bridging. Heart. 2000;84:403-08.
- [33] Marti V, Ramirez J, Lamich R, Garcia J, Guiteras P, Aymat RM, et al. Coronary stent placement for recurrent angina secondary to myocardial bridging [Spanish]. Rev Med Chil. 1998;126:1362-66.
- [34] Pratt JW, Michler RE, Pala J, Brown DA. Minimally invasive coronary artery bypass grafting for myocardial muscle bridging. Heart Surg Forum. 1999;2:250-53.
- [35] Stables RH, Knight CJ, McNeill JG, Sigwart U. Coronary stenting in the management of myocardial ischaemia caused by muscle bridging. Br Heart J. 1995;74:90-92.
 [36] Hilling AD, Margania G, Dala G, Sigwart U. Coronary stenting in the management of myocardial ischaemia caused by muscle bridging. Br Heart J. 1995;74:90-92.
- [36] Hillman ND, Mavroudis C, Backer CL, Duffy CE. Supra-arterial decompression myotomy for myocardial bridging in a child. Ann Thorac Surg. 1999;68:244-46.
 [37] Amagan G, Ordel O, Backer CL, Marking J, Children M, Chille M, Chille M, Children M, Chille M, Children M, Children M,
- [37] Atmaca Y, Ozdol C, Pamir G, Kilickap M, Oral D. Successful surgical resection of a muscular bridge in a patient with nonobstructive hypertrophic cardiomyopathy-a case report. Angiology. 2002;53:225-27.
- [38] Jeremias A, Haude M, Ge J, Gorge G, Liu F, Konorza T, et al. Emergency stent implantation in the area of extensive muscle bridging of the anterior interventricular ramus after post-interventional dissection [German]. Z Kardiol. 1997;86:367-72.
- [39] Prendergast BD, Kerr F, Starkey IR. Normalisation of abnormal coronary fractional flow reserve associated with myocardial bridging using an intracoronary stent. Heart. 2000;83:705-07.
- [40] Abhilash SP, Krishna kumar B, Velappan P, Gupata P, Viswanathan S, Geoege Koshy A. Myocardial bridging. clinical and angiographic profile in last 5 years; a study of 129 cases. Kerala Heart Journal. 2010:9-13.
- [41] Sujatha M, Subhadra Devi V, Raju CSS, Yugandhar B, Nagaraju. Angiographic aspects of myocardial bridges. Int J Anat Res. 2015;3(4):1689-96.
- [42] Jothi SDRS, Hemanth Kumar K, Rao RN, Antony J, Sai SD, SreeLekha D. Myocardial bridges over interventricular branches of coronary arteries. Jour of Med Sc & Tech. 2012;1(1):26-29.
- [43] Bourassa MG, Butanaru A, Lesparance J, Tardiff JC. Symptomatic myocardial bridges: Overview of ischaemic mechanisms and current diagnostic and treatment strategies. J Am Coll Cardiol. 2003;41:351-59.
- [44] Ishii T, Asuwa N, Masuda S, Ishikawa Y. The effects of a myocardial bridge on coronary atherosclerosis and ischemia. J Pathol. 1998;185:04-09.
- [45] Ishikawa Y, Ishii T, Asuwa N, Masuda S. Absence of atherosclerosis evolution in the coronary arterial segment covered by myocardial tissue in cholesterol-fed rabbits. Virchows Arch. 1997;430:163-71.
- [46] Boyd JH, Pargaonkar VS, Scoville DH. Surgical unroofing of hemodynamically significant left anterior descending myocardial bridges. Ann Thorac Surg. 2017;103:1443-50.
- [47] Tomasevic M, Dikic M, Ostojic M. Stenting a myocardial bridge: A wrong decision in STEMI? Acta Cardiol. 2011;66(1):89-91.

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