

Angiographic prevalence of myocardial bridging

Miyokardiyal bandın anjiyografik prevalansı

Serkan Çay, Sezgin Öztürk, Gökhan Cihan, Halil L. Kısacık, Şule Korkmaz

Department of Cardiology, Yüksek İhtisas Heart-Education and Research Hospital, Ankara, Turkey

ABSTRACT

Objective: Muscle fibers overlying the intramyocardial segment of an epicardial coronary artery are termed myocardial bridging. Variable prevalence has been described at autopsy and angiographic series with small and large sample sizes. The aim of the study was to investigate the angiographic prevalence of myocardial bridging in 25982 patients from Turkey.

Methods: We performed a retrospective study, evaluated the cases with myocardial bridging among patients undergone selective coronary angiography, and searched the angiographic prevalence of myocardial bridging in a very large sample size. We studied also the correlation between the severity of the bridging and risk factors for coronary artery disease.

Results: Among 25982 patients we found 316 cases of myocardial bridging in a retrospective manner. The total prevalence was 1.22%. Although, 96.52% of patients with myocardial bridging had the lesion in the left anterior descending coronary artery (LAD) as expected, distribution of bridges between mid- and distal segments were almost equal (52.79% and 47.21%, respectively). We sub classified patients in two groups, Group A (<50% of systolic compression) and Group B (≥50% of systolic compression), according to the amount of systolic compression of LAD and studied relationship of risk factors for coronary artery disease between groups. Another subclassification was also made for patients having myocardial bridging without coronary or valvular heart disease and hypertrophic obstructive cardiomyopathy; Group 1 (<50% of systolic compression) and Group 2 (≥50% of systolic compression). In these patients we studied correlation between the severity of the myocardial bridging and risk factors for coronary artery disease. The prevalence of bridges in circumflex and right coronary arteries individually and in all arteries as combination was also studied.

Conclusion: In a very large group of patients from Turkey undergone selective coronary artery angiography, the angiographic prevalence of myocardial bridging was slightly higher than expected. Only diabetes mellitus as a risk factor for coronary artery disease was higher in groups representing <50% of systolic compression (Group A and 1) than in groups representing ≥50% of systolic compression (Group B and 2) but the importance of this result is not known. (*Anadolu Kardiyol Derg 2006; 6: 9-12*)

Key words: Angiographic prevalence, myocardial bridging

ÖZET

Amaç: Bir epikardiyal koroner arterin intramiyokardiyal bölümünü saran kas liflerine miyokardiyal band denir. Küçük ve büyük çaplı otopsi ve anjiyografik serilerde değişik prevalanslar bildirilmiştir. Selektif koroner anjiyografi uygulanan tüm hastalar arasındaki miyokardiyal bandı olan vakaları değerlendirmek ve büyük çaplı bir seride miyokardiyal bandın anjiyografik prevalansını araştırmak amacıyla retrospektif bir çalışma düzenlendi. Ayrıca miyokardiyal bandın şiddeti ve koroner arter hastalığı risk faktörleri ile arasındaki korelasyon çalışıldı.

Yöntemler: Retrospektif olarak 25982 hasta arasında miyokardiyal band prevalansı araştırıldı.

Bulgular: Toplam 25982 hasta arasında miyokardiyal bandlı 316 vaka bulundu. Toplam prevalans %1.22 idi. Miyokardiyal band hastalarının %96.52'sinde beklenildiği gibi sol ön inen koroner arterde lezyon bulunsa da, bandların orta ve distal bölümlerdeki dağılımı hemen hemen eşitti (sırasıyla %52.9 ve %47.21). Ayrıca sol ön inen koroner arterin sistolik sıkışmasının miktarına göre hastalar Grup A (<%50 sistolik sıkışma) ve Grup B (≥%50 sistolik sıkışma) olarak iki alt gruba ayrıldı ve gruplar arasında risk faktörlerinin ilişkisi çalışıldı. Ayrıca başka bir alt sınıflandırma yapıldı; Grup 1 (<%50 sistolik sıkışma) ve Grup 2 (≥%50 sistolik sıkışma). Burada, koroner ya da valvüler kalp hastalığı ve hipertrofik obstrüktif kardiyomyopati olmayan miyokardiyal bandlı hastalarda miyokardiyal bandın şiddeti ve koroner arter hastalığı risk faktörleri ile arasındaki ilişki çalışıldı. Ayrıca sirkumfleks ve sağ koroner arterler ayrı ayrı ve kombine olarak tüm arterlerin prevalansı çalışıldı.

Sonuç: Türkiye'den selektif koroner arter anjiyografi uygulanan hastaların büyük bir grubunda miyokardiyal bandın anjiyografik prevalansı beklenenden biraz daha fazla bulunmuştur. Yalnızca koroner arter hastalığı risk faktörü olarak diyabet varlığı <%50 sistolik sıkışması bulunan gruplarda (grup A ve 1) ≥%50 sistolik sıkışması olan gruplardan (grup B ve 2) daha fazla bulunmuştur fakat bu sonucun önemi bilinmemektedir. (*Anadolu Kardiyol Derg 2006; 6: 9-12*)

Anahtar kelimeler: Anjiyografik prevalans, miyokardiyal band

Introduction

Muscle fibers overlying the intramyocardial segment of an epicardial coronary artery are termed myocardial bridging. It was first mentioned by Reyman in 1737 (1) and first described by Crainicianu in the early 1920s (2). Portmann and Iwig first reported the radiological appearance of transient stenosis in a segment of the left anterior descending coronary artery (LAD) during systole in 1960 (3). Myocardial bridging is generally thought as a harmless anatomical variant of the coronary arteries (4,5). But myocardial bridging may be associated with myocardial ischemia and infarction (6-11), coronary artery spasm (12), conduction abnormalities (13), ventricular arrhythmias (14), and sudden death (15,16). In pathological series, the prevalence has varied from 5% to 86% (17-20) (Table 1) and in angiographic series, the prevalence has been shown as being between 0.5% and 33% (10,21-26) (Table 2). Variation at angiography may in part be attributable to small and thin bridges causing little compression.

We designed a retrospective study, evaluated the prevalence of myocardial bridging in patients having selective coronary artery angiography, and studied the correlation between the severity of the myocardial bridging and risk factors for coronary artery disease.

Methods

We retrospectively evaluated the angiographic reports of patients with coronary artery disease and normal coronary arteries ($n=25982$) and searched for angiographic prevalence of myocardial bridging as totally, individually, and combinations for LAD, left circumflex (Cx), and right coronary (RCA) arteries between January 2000 and November 2004. We calculated also the prevalence of myocardial bridging in LAD segments as mid- and distal LAD, separately. Cases with myocardial bridging were classified as Group A and B for all of patients, and as Group 1 and 2 for patients with bridging and without coronary artery disease according to the percentage of systolic compression of the coronary artery. Patients in Group A and 1 had $<50\%$ of systolic compression of epicardial coronary arterial segment, and patients with $\geq 50\%$ of systolic compression represented Group B and 2. Cardiovascular risk factors were evaluated between groups. All data about angiographic analysis were obtained from files of the patients electronically with computer analysis. Only in the patients with cardiac problems and without selective coronary artery angiography (congestive heart failure, myocarditis, heart rhythm abnormalities, pericardial diseases, etc.) were not included in the study during 4-year period ($n=10485$).

Statistical analysis

Data were analyzed with the SPSS software version 10.0 for Windows. Continuous variables from the study groups were reported as mean \pm standard deviation, categorical variables as percentages. Differences in baseline characteristics between groups were assessed with t tests for continuous variables and χ^2 tests for binary variables. All tests were two-sided with a 0.05 significance level.

Results

The total number of the patients with selective coronary angiographic analysis (ample size, n) was 25982 and the total number of the cases with myocardial bridging was 316. Thus the prevalen-

ce was 1.22% (316 of 25982) totally. Their ages ranged from 21 to 86 years (mean 55.6 ± 11.3) and 82% (259 of 316) of patients was male. Female patients were significantly older than male ones (58.9 ± 9.8 years and 54.9 ± 11.4 years, respectively, $p=0.015$). Among these patients 146 patients had coronary artery disease (CAD) and myocardial bridging, 21 had valvular heart disease and myocardial bridging without coronary artery disease, 1 had hypertrophic obstructive cardiomyopathy and myocardial bridging without coronary artery disease, and the remaining 148 patients had myocardial bridging without coronary artery disease. Among patients with myocardial bridging 37.7% had hypertension, 12.7% had diabetes mellitus, 42.7% had hyperlipidemia, 29.4% had family history, and smoking was present in 41.8% of patients. Two groups were constituted according to the percentage of systolic reduction of the epicardial coronary artery lumen: Group A, $\geq 50\%$ (182 patients) and Group B, $\geq 50\%$ (134 patients). There was no significant difference between two groups in age, hypertension, hyperlipidemia, family history, and smoking except diabetes (Table 3). Among patients with bridging and without coronary artery disease ($n=148$), two subgroups were also identified according to the percentage of systolic reduction of the epicardial coronary artery lumen: Group 1, $\geq 50\%$ (97 patients) and Group 2, $\geq 50\%$ (51 patients). There was also no significant difference between these two subgroups in age, hypertension, hyperlipidemia, family history, and smoking except diabetes (Table 4). The rate of coexisting coronary artery disease in diabetics and in those without were 23/40 (57.5%) and 123/276 (44.6%), respectively. A higher rate of CAD in subjects with diabetes was found as expected.

The prevalence was shown as being between 0.96% and 1.68% per year (Table 5). Among all of the patients 96.52% (305 of 316) constituted the prevalence of LAD bridges (1.17% [305 of 25982] of the total number). The prevalence of bridging of mid-LAD and distal LAD myocardial bridging were also 52.79% (161 of 305) and 47.21% (144 of 305), respectively (Table 6). The prevalence of bridging in Cx and RCA was 2.22% (7 of 316) and 0.63% (2 of 316), respectively (0.03% [7 of 25982] and 0.08% [2 of 25982] of the total number, respectively). The prevalence of myocardial bridging in the LAD and Cx coronary arteries was 0.63% (2 of 316) of bridging cases and 0.08% (2 of 25982) of total ones, simultaneously. We found no patient having myocardial bridging in the LAD and RCA, Cx and RCA, and LAD, Cx, and RCA coronary arteries, simultaneously (Table 7).

Discussion

Although myocardial bridges are most commonly found in the middle segment of the LAD coronary artery some cases of Cx and RCA myocardial bridges have been reported in the literature (8,27-30). Two types of myocardial bridging have been reported as superficial bridges crossing the coronary artery perpendicularly or at an acute angle toward the apex forming 75% of cases, and muscle fibers arising from the right ventricular apex that cross the LAD transversely or obliquely forming 25% of cases (20). Most of the myocardial bridges seen in autopsy series are not seen angiographically. Variation at angiography may in part be attributable to small and thin bridges causing little compression because only the deep type of myocardial bridges can be seen angiographically (20). A high prevalence has also been reported in patients with hypertrophic obstructive cardiomyopathy (HOCM) (31). In our study, only one patient had myocardial bridge among patients with HOCM thus the prevalence was 7.7% ($n=13$, small sample size).

The gold standard diagnostic tool for diagnosing myocardial bridge is selective coronary artery angiography. But the new imaging techniques like intravascular ultrasound (IVUS), intracoronary Doppler ultrasound (ICD), and intracoronary pressure devices as invasive techniques (32, 33) and electron beam tomography (EBT), multislice CT (MSCT), magnetic resonance tomography (MRT), or transthoracic Doppler echocardiography as noninvasive imaging techniques (34) can be used for diagnosis of functional and morphological status of bridges.

In our study, only diabetes mellitus as a risk factor for coronary artery disease was higher in group A and 1 than in group B and 2 but the importance of this result is not known. The potential impact of vasoreactivity in myocardial bridging might be the reason. Shear stress alteration in myocardial bridging might be resulted in endothelial dysfunction. Increased intracoronary pressure is also associated with impairment of endothelium-dependent vasorelaxation. Increased vasoconstriction and decreased coronary blood flow to acetylcholine in patients with myocardial bridging have been demonstrated by Herrmann et al. (35). Decreased vasodilatation to nitroglycerine has also been shown (35). Addition of these factors to structural coronary lumen compression

might exaggerate the severity of narrowing and be resulted in clinical complications. Endothelial dysfunction and so vasoreactivity are also seen in diabetes. Thus, additive effect may be present in the presence of diabetes. Atherosclerotic plaque generally is fo-

Table 4. Baseline characteristics and cardiovascular risk factors of patients in two sub groups

	Group 1 (n=97)	Group 2 (n=51)	p
Age, years	53.31±11.05	52.86±12.35	NS
Sex (male/female)	67/30	42/9	NS
Hypertension, n (%)	39/97 (40.2)	15/51 (29.4)	NS
Diabetes mellitus, n (%)	15/97 (15.5)	1/51 (2.0)	0.012
Hyperlipidemia, n (%)	42/97 (43.3)	20/51 (39.2)	NS
Family history, n (%)	35/97 (36.1)	15/51 (29.4)	NS
Smoking history, n (%)	32/97 (33.0)	16/51 (31.4)	NS

Group 1, the percentage of systolic compression of LAD coronary artery lumen < 50%
Group 2, the percentage of systolic compression of LAD coronary artery lumen ≥ 50%
CAD - coronary artery disease, ECG - electrocardiography, NS - nonsignificant

Table 1. Autopsy prevalence of bridging cases in previous studies

Authors (Reference No.)	Sample size, n	%
Geiringer (17)	100	23
Edwards, et al (18)	276	5
Poláček, et al (19)	70	86
Ferreira, et al (20)	90	56

Table 2. Angiographic prevalence of bridging cases in previous studies

Authors (Reference No.)	Sample size, n	%
Noble, et al (21)	5250	0.5
Ishimori, et al (22)	313	1.6
Greenspan, et al (23)	1600	0.9
Rossi, et al (10)	1146	4.5
Kramer, et al (24)	658	12
Wymore, et al (25)	64	33
Juilliére, et al (26)	7467	0.8

Table 3. Baseline characteristics and cardiovascular risk factors of patients in two groups.

	Group A (n=182)	Group B (n=134)	p
Age, years	55.7±10.7	55.6±12.0	NS
Sex, (male/female)	143/39	116/18	NS
Hypertension, n (%)	74/182 (40.7)	45/134 (33.6)	NS
Diabetes mellitus, n (%)	29/182 (15.9)	11/134 (8.2)	0.041
Hyperlipidemia, n (%)	77/182 (42.3)	58/134 (43.3)	NS
Family history, n (%)	56/182 (30.8)	37/134 (27.6)	NS
Smoking history, n (%)	70/182 (38.5)	62/134 (46.3)	NS
The presence of CAD, n (%)	76/182 (41.8)	70/134 (52.2)	0.031

Group A, the percentage of systolic compression of LAD coronary artery lumen < 50%
Group B, the percentage of systolic compression of LAD coronary artery lumen ≥ 50%
CAD - coronary artery disease, ECG - electrocardiography, NS - nonsignificant

Table 5. Prevalence of myocardial bridging per year

Time course	Selective CAG, n	Myocardial bridging, n	Prevalence, %
Year 2000	5838	56	0.96
Year 2001	4975	49	0.98
Year 2002	5571	66	1.18
Year 2003	6204	104	1.68
Year 2004	3394	41	1.21
Total	25982	316	1,22

CAG - coronary angiography

Table 6. Prevalence of LAD myocardial bridging according to segment involvement

LAD segment involvement	Myocardial bridging, n	Prevalence, %, (n=305)
Mid-LAD	161	52.79
Distal LAD	144	47.21

LAD - left anterior descending coronary artery

Table 7. Angiographic prevalence of bridging cases in our study

Coronary artery involvement	Myocardial bridging	Prevalence, % cases, (n=316)	Prevalence, % (n=25982)
LAD only	305	96.52	1.17
Cx only	7	2.22	0.03
RCA only	2	0.63	0.008
LAD and Cx	2	0.63	0.008
LAD and RCA	0	0	0
Cx and RCA	0	0	0
LAD, Cx, and RCA	0	0	0
Total	316	100	1.22

Cx - circumflex coronary artery; LAD - left anterior descending coronary artery; RCA - right coronary artery

und proximal to the bridge; however the segment under the bridge is spared. Ischemia can be explained neither by that atherosclerotic segment nor by systolic compression alone. Some functional findings determined by intravascular ultrasound and Doppler can explain the mechanism; a specific echolucent half moon phenomenon around the bridge segment, systolic compression of the bridge segment, accelerated flow velocity at early diastole, reduced antegrade systolic flow or retrograde systolic flow in the proximal segment, and reduced diastolic/systolic velocity ratio (32).

As mentioned above, for LAD myocardial bridges, the pathology is generally found in the middle portion of the LAD coronary artery. However we found that, bridges of LAD coronary artery were distributed almost equally between the middle and distal segments and no myocardial bridging was present in the proximal segment, interestingly. This equality is probably due to segment definition; the segment between first diagonal and second diagonal coronary arteries is termed middle segment and the segment after second diagonal branch is termed distal one in our study.

In conclusion, angiographic prevalence of myocardial bridging in our study is slightly higher than the results of other studies having big sample size. We found also that, the prevalence of mid and distal LAD myocardial bridges were almost similar that is in contrary to general conviction and no relationship was observed between studied groups for coronary risk factors except diabetes.

Study limitations

The limitation of the study is that angiograms were not reviewed; just the written reports were studied. Bridging is more likely to be noted after intra-coronary nitroglycerin. But, all of the patients did not receive intra-coronary nitroglycerin at the time of angiography in our study. Thus the prevalence is likely underestimated because: 1) nitroglycerine was not given and 2) the article is retrospective in nature. Angiographers were likely not specifically trained to identify myocardial bridges and subtle bridges might have been missed.

References

1. Reyman HC. Disertatio de vasis cordis propriis (dissertation). Göttingen: Med Diss Univ. 1737.
2. Cranicianu A. Anatomische Studien über die Coronararterien und experimentelle Untersuchungen über ihre Durchgängigkeit. Virchows Arch A Pathol Anat 1922; 238: 1-8.
3. Portmann W, Iwig J. Die intramurale Koronarie im Angiogramm. Fortschr Röntgenstr 1960;92:129-32.
4. Schulte MA, Waller BF, Hull MT, Pless JE. Origin of the left anterior descending coronary artery from the right aortic sinus with intramyocardial tunneling to the left side of the heart via the ventricular septum: A case against clinical and morphologic significance of myocardial bridging. Am Heart J 1985;110:499-501.
5. Visscher DW, Miles BL, Waller BF. Tunneled ('bridged') left anterior descending coronary artery in a newborn without clinical or morphologic evidence of myocardial ischemia. Cathet Cardiovasc Diagn 1983;9:493-6.
6. Feldman AM, Baughman KL. Myocardial infarction associated with a myocardial bridge. Am Heart J 1986;111:784-7.
7. Yano K, Yoshino H, Taniuchi M, Kachi E, Shimizu H, Watanuki A, et al. Myocardial bridging of the left anterior descending coronary artery in acute inferior wall myocardial infarction. Clin Cardiol 2001; 24: 202-8.
8. Arjomand H, AlSalman J, Azain J, Amin D. Myocardial bridging of left circumflex coronary artery associated with acute myocardial infarction. J Invasive Cardiol 2000;12: 431-4.
9. Ortega-Carnicer J, Fernandez-Medina V. Impending acute myocardial infarction during severe exercise associated with a myocardial bridge. J Electrocardiol 1999; 32: 285-8.
10. Rossi L, Dander B, Nidasio GP, Arbustini E, Paris B, Vassanelli C, et al. Myocardial bridges and ischemic heart disease. Eur Heart J 1980; 1: 239-45.
11. Furniss SS, Williams DO, McGregor CG. Systolic coronary occlusion due to myocardial bridging - a rare cause of ischemia. Int J Cardiol 1990; 26: 116-7.
12. Ciampicotti R, El Gamal M. Vasospastic coronary occlusion associated with a myocardial bridge. Cathet Cardiovasc Diagn 1988; 14: 118-20.
13. den Dulk K, Brugada P, Braat S, Hedde B, Wellens HJ. Myocardial bridging as a cause of paroxysmal A-V block. J Am Coll Cardiol 1983; 1: 965-9.
14. Feld H, Guadanino V, Hollander G, Greengart A, Lichstein E, Shani J. Exercise-induced ventricular tachycardia in association with a myocardial bridge. Chest 1991; 99: 1295-6.
15. Morales AR, Romanelli R, Boucek RJ. The mural left anterior descending coronary artery, strenuous exercise and sudden death. Circulation 1980; 62: 230-7.
16. Cutler D, Wallace JM. Myocardial bridging in a young patient with sudden death. Clin Cardiol 1997;20:581-3.
17. Geiringer E. The mural coronary. Am Heart J 1951;41:359-68.
18. Burnsides C, Edwards JC, Lansing AI, Swarm RL. Arteriosclerosis in the intramural and extramural portions of coronary arteries in the human heart. Circulation 1956;13:235-41.
19. Poláček P, Kralove H. Relation of myocardial bridges and loops on the coronary arteries to coronary occlusions. Am Heart J 1961;61:44-52.
20. Ferreira AG Jr, Trotter SE, König B Jr, Decourt LV, Fox K, Olsen EG. Myocardial bridges: morphological and functional aspects. Br Heart J 1991;66:364-7.
21. Noble J, Bourassa MG, Petitclerc R, Dyrda I. Myocardial bridging and milking effect of the left anterior descending coronary artery: normal variant or obstruction? Am J Cardiol 1976;37:993-9.
22. Ishimori T. Myocardial bridges: a new horizon in the evaluation of ischemic heart disease. Cath Cardiovasc Diagn 1980;6:355-7.
23. Greenspan M, Iskandrian AS, Catherwood E, Kimbiris D, Bemis CE, Segal BL. Myocardial bridging of the LAD: evaluation using exercise thallium-201 myocardial scintigraphy. Cathet Cardiovasc Diagn 1980;6:173-80.
24. Kramer JR, Kitazume H, Proudfit WL, Sones FM Jr. Clinical significance of isolated coronary bridges: benign and frequent condition involving the left anterior descending artery. Am Heart J 1982;103:283-8.
25. Wymore P, Yedlicka JW, Garcia-Medina V, Olivari MT, Hunter DW, Castaneda-Zuniga WR, et al. The incidence of myocardial bridges in heart transplants. Cardiovasc Intervent Radiol 1989;12:202-6.
26. Juillièrre Y, Berder V, Suty-Selton C, Buffet P, Danchin N, Cherrier F. Isolated myocardial bridges with angiographic milking of left anterior descending coronary artery: a long-term follow-up study. Am Heart J 1995;129:663-5.
27. Angellini P, Leachman R, Autrey A. Atypical phasic coronary artery narrowing. Cathet Cardiovasc Diagn 1986;12:39-43.
28. Gurewitch J, Gotsman MS, Rozenman Y. Right ventricular myocardial bridge in a patient with pulmonary hypertension. A case report. Angiology 1999;50:345-7.
29. Woldow AB, Goldstein S, Yazdanfar S. Angiographic evidence of right coronary bridging. Cathet Cardiovasc Diagn 1994;32:351-3.
30. Garg S, Brodison A, Chauhan A. Occlusive systolic bridging of circumflex artery. Catheter Cardiovasc Interv 2000;51:477-8.
31. Achrafi H. Hypertrophic cardiomyopathy and myocardial bridging. Int J Cardiol 1992;37:111-2.
32. Ge J, Jeremias A, Rupp A, Abels M, Baumgart D, Liu F, et al. New signs characteristic of myocardial bridging demonstrated by intracoronary ultrasound and Doppler. Eur Heart J 1999;20:1707-16.
33. Bourassa MG, Butnaru A, Lespérance J, Tardif JC. Symptomatic myocardial bridges: overview of ischemic mechanisms and current diagnostic and treatment strategies. J Am Coll Cardiol 2003;41:351-9.
34. Möhlenkamp S, Hort W, Ge J, Erbel R. Update on myocardial bridging. Circulation 2002;106:2616-22.
35. Herrmann J, Higano ST, Lenon RJ, Rihal CS, Lerman A. Myocardial bridging is associated with alteration in coronary vasoreactivity. Eur Heart J 2004;25:2134-42.