Myocardial bridge: a bridge to atherosclerosis

Miyokardiyal köprüleme: Ateroskleroza köprü

Hamza Duygu, Mehdi Zoghi, Sanem Nalbantgil, Bahadır Kırılmaz, Uğur Türk, Filiz Özerkan, Azem Akıllı, Mustafa Akın

Department of Cardiology, Medical Faculty, Ege University, İzmir, Turkey

ABSTRACT

Objective: Myocardial bridge (MB) is a congenital anomaly characterized by narrowing during systole of some of the epicardial coronary arterial segments running in the myocardium. Although, it is considered as a benign anomaly, it may lead to such complications as acute myocardial infarction, ventricular tachycardia, syncope, atrioventricular block and sudden cardiac death. In this study, we aimed to investigate demographic, clinical and angiographic characteristics of the patients with MB found on coronary angiography.

Methods: The present study included 71 patients with MB found on coronary angiographies performed in our institution between January 1999 and September 2003. Based on the findings on angiography, the patients were subdivided into group A (n=41) and group B (n=30). The patients in the group A had no atherosclerotic lesion and the patients in the group B had coronary artery disease in addition to MB. Angiographic, demographic and clinical characteristics of both groups were compared.

Results: There were no differences between two groups in distribution of gender and risk factors of coronary artery disease whereas mean age of the patients in the group A was lower (47±5 years vs 55±11 years, p=0.01). Frequency of two or more risk factors for coronary artery disease in a particular patient was significantly higher in the group B (55% vs 30%, p=0.03). Myocardial bridge was located at proximal or mid segments of left anterior descending artery (LAD) in 40 patients whereas its presence in both LAD and right coronary artery was found only in one patient in group A. Mean bridging percent was 43±27% in group A. Localization of MB was LAD in 29 patients of group B. One patient with severe aortic valve stenosis in this group had MB at first septal branch. Mean bridging percent was 70±25% in group B, which was significantly higher than in group A (p<0.05). Atherosclerotic narrowing developed in only LAD in 14 patients, LAD and other vessels in 7 patients and in the vessels without MB in 9 patients. In patients with MB in LAD atherosclerotic narrowing of vessel developed proximally to the MB. Clinically, stable angina pectoris was seen more frequently in group A than group B (70% vs 35%, p=0.01), whereas the frequency of acute coronary syndrome was higher in group B (65% vs 30%, p=0.04). In regard to therapeutic approach, more patients in the group A received medical management (80% vs 50%, p=0.01), while more patients in the group B underwent surgical and percutaneous interventions (50% vs 18%, p=0.04).

Conclusion: Myocardial bridge probability should be considered in young patients presenting with angina or if the same symptoms are persistent in the patients without more than one risk factor for coronary artery disease. Myocardial bridge may initiate the development of atherosclerotic lesion or may facilitate progression of atherosclerosis in the proximal segment of the vessel. The risk of acute coronary syndrome rises when atherosclerosis is superimposed on MB. Myocardial bridge should be considered in the young patients, presenting with angina or its equivalents without atherosclerotic lesions on coronary angiography. *(Anadolu Kardiyol Derg 2007; 7: 12-6)*

Key words: Myocardial bridge, atherosclerosis, coronary angiography

ÖZET

Amaç: Miyokardiyal köprüleme (MB), epikardiyal arterlerin bazı bölümlerinin intramiyokardiyal seyretmesi ile karakterize konjenital bir anomalidir. Benign bir anomali olmakla birlikte akut miyokard infarktüsü, ventrikül taşikardisi, senkop, atriyoventriküler blok ve ani kardiyak ölüm gibi komplikasyonlara yol açabilir. Bu çalışmada koroner anjiyografide MB saptanan hastaların demografik, klinik ve anjiyografik özelliklerini araştırmayı amaçladık.

Yöntemler: Çalışmaya Ocak 1999-Eylül 2003 tarihleri arasında kliniğimizde yapılan koroner anjiyografilerde MB saptanan 71 hasta alındı. Anjiyografik bulgulara göre hastalar grup A (n=41) ve grup B (n=30) olmak üzere iki alt gruba ayrıldı. Grup A aterosklerotik lezyon saptanmayan, grup B ise MB'e ek olarak koroner arter hastalığı saptanan hastalardan oluşmaktaydı. İki grubun anjiyografik, demografik ve klinik özellikleri karşılaştırıldı.

Bulgular: İki grup arasında cinsiyet ve koroner arter hastalığı risk faktörleri açısından fark saptanmazken A grubundaki hastaların yaş ortalaması daha düşüktü (47±5'e karşılık 55±11 yıl, p=0.01). Aynı hastada iki veya daha fazla koroner arter hastalığı risk faktörü sıklığı B grubunda anlamlı olarak daha fazla bulundu (%55'e karşılık %30, p=0.03). Miyokardiyal köprüleme grup A'da, sadece bir hastada sol ön inen (LAD) ve sağ koroner arterlerde, 40 hastada ise proksimal veya mid LAD'de yerleşmekteydi. Grup A'da ortalama bridge yüzdesi %43±27 idi. Grup B'de MB, ciddi aortik kapak darlığı olan bir hastada birinci septal dalda, 29 hastada ise LAD'de yerleşmekteydi. Grup B'de ortalama bridge yüzdesi %70±25 idi (p<0.05). Aterosklerotik daralma 14 hastada sadece LAD'de, 7 hastada LAD ve diğer damarlarda ve 9 hastada ise MB dışındaki damarlarda saptandı. LAD'de MB'e ek olarak aterosklerotik darlık bulunan hastaların hepsinde ateroskleroz MB proksimalinde yerleşmekteydi. Klinik olarak, akut koroner sendrom sıklığı grup B'de daha fazla iken (%65'e karşılık %30, p=0.04), stabil anjina pektoris grup A'da grup B'den daha fazla idi (%70'e karşılık %35, p=0.01). Tedavi yaklaşımı olarak, grup A'da daha çok tıbbi tedavi uygulanırken (%80'e karşılık %50, p=0.01), grup B'de daha çok cerrahi ve girişimsel yaklaşımlar uygulandı (%50'e karşılık %18, p=0.04).

Sonuç: Anjina ile başvuran genç hastalarda veya birden fazla koroner arter hastalığı risk faktörü olmayan hastalarda semptomlar devam ediyorsa MB olasılığı dikkate alınmalıdır. Miyokardiyal köprüleme damarın proksimal bölümünde aterosklerotik lezyon gelişimini başlatabilir veya ilerlemesini hızlandırabilir. Miyokardiyal köprülemeye ateroskleroz eklendiğinde akut koroner sendrom riski artar. Koroner anjiyografilerde aterosklerotik lezyon bulunmayan genç hastalarda MB olasılığına dikkat edilmelidir. *(Anadolu Kardiyol Derg 2007; 7: 12-6)*

Anahtar kelimeler: Miyokardiyal köprüleme, ateroskleroz, koroner anjiyografi

Address for Correspondence: Hamza Duygu, MD, Ege University Medical Faculty, Cardiology Department, Bornova, 35100, İzmir, Turkey
Tel.: +90 232 390 40 01 Fax: +90 232 390 32 87 E-mail: hamzakard@yahoo.com

Introduction

Myocardial bridge (MB) is an anatomical variation characterized by narrowing during systole of some of the epicardial coronary arterial segments running in the myocardium. It can be encountered in 0.5 to 16% of routine coronary angiographies (1). Although, it is considered as a benign anomaly it may lead to such complications as acute myocardial infarction, ventricular tachycardia, syncope, atrioventricular blocks and sudden cardiac death (2,3). The present study investigated the clinical, demographic and angiographic characteristics of patients with MB.

Methods

The present study included 71 patients who had positive exercise testing or myocardial ischemia on thallium scintigraphy and 'milking effect' from 6272 coronary angiographies performed in our institution between January 1999 and September 2003. Angiographic, demographic and clinic characteristics of the patients were reviewed retrospectively from their medical records. Risk factors for coronary artery disease (hypertension, diabetes mellitus, smoking, dyslipidemia and family history) and mode of presentation to the hospital (stable angina pectoris, unstable angina pectoris and acute myocardial infarction), treatment modality applied and medication given on the time of discharge from the hospital were examined.

Coronary angiography was performed by standard Judkins' technique with a biplane cineangiography system. The presence of MB was defined as the following criteria: narrowing of coronary vessel lumen during systole and dilation during diastole; no evidence for occurrence of coronary vasospasm. Location of the MB, narrowing rate and associated atherosclerotic narrowing were reviewed on coronary angiography. For the quantitative assessment of the severity of MB, we used a computer-assisted data analyzing system for angiography (DCI, Philips Medical, version 3.3, The Netherlands). Angiographically determined percent narrowing of the MB was calculated as {(end-diastolic diameter - end-systolic diameter)/end-diastolic diameter}x 100 (4). Based on the findings on coronary angiography, the patients were subdivided into two subgroups: Group A (n=41) with no atherosclerotic lesion and Group B (n=30) with atherosclerotic coronary artery disease in addition to MB. Angiographic, demographic and clinical characteristics of both groups were compared. Angiographically documented coronary artery disease was defined as stenosis of more than >50%. The diagnosis of myocardial infarction required the presence of at least 2 of the following criteria (5): a history of characteristic prolonged (≥ 30 min) pain or discomfort (6); creatine kinase (CK) elevation exceeding twice the upper limit of normal (or CK-MB ≥ 50% of total CK) (7); presence of new Q waves or new abnormal ST-T segment features. Unstable angina was defined as pain at rest with at least 2 episodes during the previous 48 h, at least one of which lasted ≥ 20 min, or ST-segment deviations that were diagnostic of myocardial ischemia during anginal attack, with no elevation of CK-MB or lactate dehydrogenase on admission to the hospital (8). Chronic stable angina was defined as effort angina with objective ischemic evidence and stable ischemic threshold over the past 6 months.

Statistical Analysis

Quantitative data were expressed as mean±standard deviation (SD) and qualitative data as percentage values. Statistical analyses were done using the software SPSS for Windows (version 10.0). Categorical data between two groups were compared with Fisher's exact test and Chi-square test and continuous variables - with Student`s unpaired t test. P values less than 0.05 were considered to be statistically significant. Pearson's correlations were used to confirm the relationship between the variables.

Results

Demographic, clinic, angiographic and treatment characteristics of both groups are seen in table 1 and 2. The mean age of the patients in the study was 51±10 years with 77% of them were male (n=51) and 23% (n=20) were female. The frequency of MB on coronary angiography was found to be 0.01%. Only MB was found in 41 patients (group A, Fig. 1) whereas 30 patients were found to have MB with atherosclerotic narrowing (group B, Fig. 2). There was no difference between two groups in distribution of

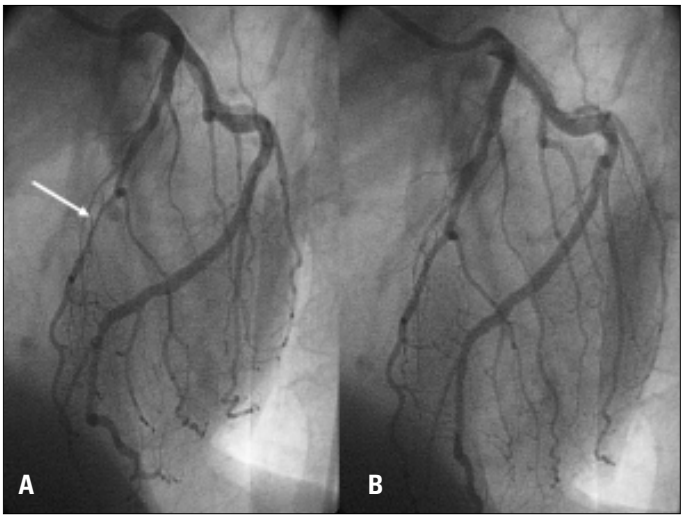

Figure 1. Coronary angiogram with myocardial bridging of the left anterior descending coronary artery during systole (A) and diastole (B)

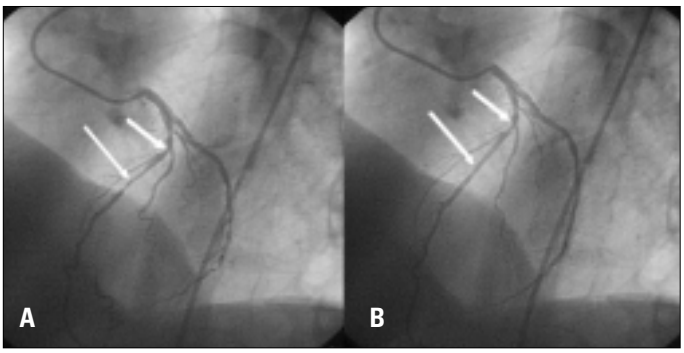

Figure 2. Typical systolic compression (big arrow) of the mid left anterior descending coronary artery (A). Diastolic lumen dimensions are normal (B). Left anterior descending coronary artery shows angiographic signs of coronary atherosclerosis (small arrow) proximal to myocardial bridge

gender and risk factors of coronary artery disease whereas mean age of the patients in the group A was lower (p=0.01) (Table 1). Frequency of two or more risk factors for coronary artery disease in a particular patient was significantly higher in the group B (p=0.03) (Table 1). Clinically, stable angina pectoris was seen more frequently in the patients in group A than in the patients in group B (p=0.01), whereas the frequency of acute coronary syndrome was higher in group B (p=0.04) (Table 2). Angina complaint was considered as atypical in 60% of the patients presented with SAP in group A. Myocardial bridge was located at proximal or mid segment of LAD in 40 patients whereas it was found at both LAD and RCA in only one patient in group A. The length of MB on angiography was 24±5 mm, the percentage of systolic narrowing was 43±27% (Table 1). There was no correlation between percentage of systolic narrowing and clinical presentation (R=0.270, p>0.05). Exercise testing was negative in half of 23 patients with mild (narrowing percentage lower than 30%) MB. Revascularization therapy was applied to patients with persistent symptoms despite medical therapy and objective evidence of ischemia in thallium scintigraphy. Surgical revascularization was made in patients with long and deep MB. Surgical treatment was advised to 6 of 11 patients with narrowing of 70% and more during systole whereas 2 and 3 of these 11 patients were advised to have stent implantation and medical follow-up, respectively. In regard to therapeutic approach, more patients in the group A received medical management while more patients the group B underwent surgical and percutaneous interventions (Table 2).

In group B MB was localized at LAD in 29 patients whereas in the first septal branch in a patient with severe aortic valve stenosis. The length of MB on angiography was 26±6 mm (compared with group A - p>0.05, Table 1), the percentage of systolic narrowing was 70±25% (compared with group A - p=0.01, Table 1). There was a significant correlation between percentage of systolic narrowing and severity of clinical presentation in group B (R=0.450, p=0.01). Atherosclerotic narrowing developed in only LAD in 14 patients, in LAD and other vessels in 7 patients and in the vessels without MB in 9 patients. Atherosclerotic lesions showed one-vessel involvement in 45% of the patients, while the remaining had two or three-vessel involvement. Eighty-five percent of the atherosclerotic narrowings were the lesions producing stenosis of 70% or more. Interestingly, atherosclerosis located proximally to the MB in all of the patients with MB concomitant atherosclerotic stenosis in the LAD and there was also positive correlation between percentage of atherosclerotic stenosis and systolic narrowing (R=0.380, p=0.03, Fig. 3).

Table 1. The comparison of demographic, angiographic characteristics and risk factors of both groups

Variables	Group A (n=41)	Group B (n=30)	p
Age, years	47±5	55±11	0.01
Gender, male/female	30/11	21/9	NS
Length of MB, mm	24±5	26±6	NS
Systolic narrowing, %	43±27	70±25	0.01
Hypertension, n (%)	14(34)	10(33)	NS
Diabetes mellitus, n (%)	2(5)	1(6)	NS
Dyslipidemia, n (%)	14(34)	12(40)	NS
Family history of CAD, n (%)	10(24)	8(26)	NS
Smoking, n (%)	18(44)	14(46)	NS
Number of more than (%) one risk factors, n	12(30)	17(55)	0.03

CAD- coronary artery disease, MB- myocardial bridge, NS- nonsignificant

Table 2. The comparison of clinical characteristics and treatment modalities of both groups

Variables	Group A	Group B	p
Stable angina pectoris, n (%)	29(70)	10(35)	0.01
Unstable angina pectoris, n (%)	8(20)	14(45)	NS
Acute myocardial infarction, n (%)	4(10)	6(20)	NS
Acute coronary syndrome, n (%)	12(30)	20(65)	0.04
Medical treatment, n (%)	33(80)	15(50)	0.01
Surgical treatment, n (%)	6(14)	9(30)	NS
Stent implantation, n (%)	2(4)	6(20)	NS
Surgical and interventional therapy, n (%)	8(18)	15(50)	0.04

NS- nonsignificant

Discussion

Muscle overlying the intramyocardial segment of an epicardial coronary artery, is termed a myocardial bridge, and the artery coursing within the myocardium is called a tunneled artery. The estimated frequency that has been reported varies from 0.5 to 16% when assessed by coronary angiography, but in some autopsy series, it is as high as 80% (9). Angiographic prevalence of MB was reported as 1.22% among almost 26.000 Turkish patients undergoing coronary angiography (10). But, the frequency of MB

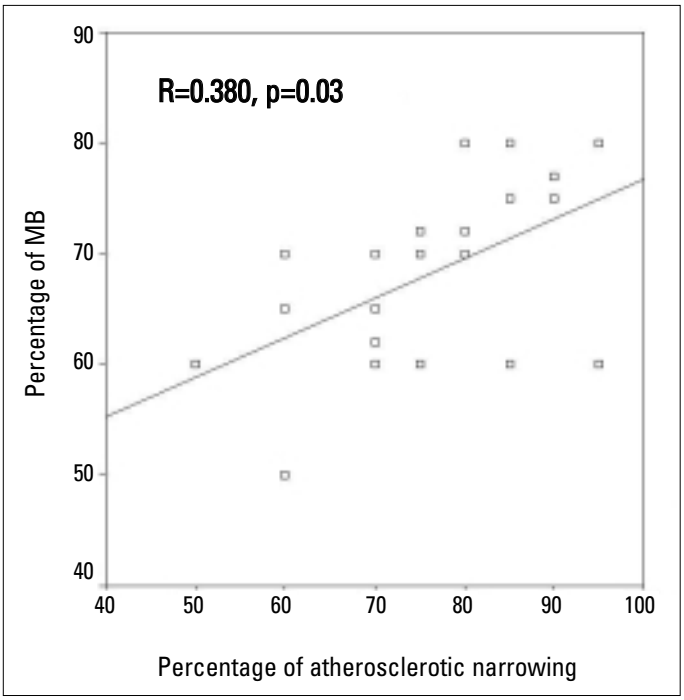

Figure 3. Relation between the percentage of myocardial bridge (MB) and percentage of atherosclerotic lesion in the left anterior descending coronary artery
MB- myocardial bridge

on coronary angiography was found to be 0.01% in our study. The cause of low frequency may be overlooking of mild MB. The current gold standard for diagnosing MB is coronary angiography with the typical ''milking effect''. But, the mild MBs may be overlooked. In uncertain cases, systolic narrowing at the myocardial bridge can be accentuated by intracoronary injection of nitroglycerin (11). With the use of intravascular ultrasound, intracoronary Doppler ultrasound and intracoronary pressure devices, morphological and functional features of myocardial bridging can be visualized and quantified (12).

Myocardial bridging is generally confined to the mid left anterior descending artery (13). In our study the 69 patients had myocardial bridges in proximal or mid LAD. But, MB in the proximal LAD was not reported in an angiographic prevalence study from Turkey (10). In addition, one case with aortic valve stenosis had septal branch and it was located in both LAD and RCA in only one patient. A high prevalence has also been reported in heart transplant recipients and in patients with hypertrophic obstructive cardiomyopathy (14). Traditionally, myocardial bridging has been considered as a benign condition, however it may lead to such complications as acute myocardial infarction, ventricular tachycardia, syncope, atrioventricular block and sudden cardiac death (15-18). In our study, 12 patients with MB with no atherosclerotic lesion presented with acute coronary syndrome.

The diagnosis of clinically important myocardial bridging should be considered in patients who have angina and do not have the traditional risk factors for coronary artery disease and the evidence of ischemia (19). Myocardial bridge has been shown to lead to ischemic heart disease by several mechanisms. One of these mechanisms is that MB increases the tendency for occurrence of atherosclerosis. Myocardial bridge causes atherosclerosis proximal to the bridge with its shear stress effect (12, 20), because, endothelium within the MB has a structure more resistant to atherosclerosis (21). We also showed endothelial dysfunction in patients with MB using flow-mediated dilatation technique in the brachial artery in another study (22).

We found that atherosclerotic lesions were located proximally to the MB in all of the patients with MB and concomitant atherosclerotic stenosis in the LAD. Ge et al (12) showed plaque formation proximal to the bridge in 90% of patients by intracoronary ultrasound. But, we evaluated atherosclerosis with coronary angiography and didn't use intracoronary ultrasound in all patients. Ishikawa et al (23) demonstrated that atherosclerosis ratio in the proximal segments of LAD (up to 2.5 cm away from the ostium) was significantly lower in cases with MB located within 5 cm from the left coronary ostium than in the corresponding segments of control group in an autopsy study. On the other hand, our study showed that atherosclerosis ratio in the proximal LAD segments up to 2.5 cm away from the ostium in distal MB location group (over 5.0 cm from the left coronary ostium) was not different from that of control group. But, we didn't evaluate atherosclerosis according to MB location from the left coronary ostium.

Myocardial bridge probability should be considered in young individuals presenting with angina and its equivalent or if the same symptoms are persistent in the patients without any risk factor for coronary artery disease or with only one risk factor. Positive exercise testing is rare in patients with MB (24). In this study, it has been shown that exercise testing may be normal especially if the MB systolic compression percentage is low.

This study has shown that with increasing number of risk factors atherosclerotic lesions developed in addition to MB and these lesions mostly localized proximal to the bridge. While patients who had only MB presented with stable angina, patients with atherosclerotic occlusion in addition to MB had high prevalence of acute coronary syndrome on admission.

We also demonstrated that patients with MB and concomitant atherosclerotic lesions (group B) more often were referred to surgery and invasive treatment approaches because of additional atherosclerotic occlusions. Klues and Haager (25, 26) had successful results with stent implantation in MB patients. In our study, 8 patients also had successful stent implantations.

In conclusion, MB should be considered in the patients with no atherosclerotic lesions on diagnostic coronary angiography. Myocardial bridge probability should be considered in young individuals presenting with angina and its equivalent or if symptoms are persistent in the patients without any risk factor for coronary artery disease or with only one risk factor. Classical noninvasive stress tests are more likely to yield negative results if systolic narrowing rate of MB is low and thus it may be difficult to establish the diagnosis. Coronary angiography is diagnostic in these cases. Myocardial bridge may initiate the development of atherosclerotic lesion or may facilitate progression by increasing shear stress on the proximal segment. The incidence of acute coronary syndrome rises in the patients when atherosclerosis is superimposed on MB. Stent implantation or surgical intervention should be considered in the patients with refractory to the treatment symptoms and systolic narrowing of coronary segment is hemodynamically significant.

Limitations of the Study

The main limitation of the study is that coronary angiographies were not reviewed. The cause of low frequency may be overlooking of mild MB. The current gold standard for diagnosing MB is coronary angiography with the typical ''milking effect''. But, the mild MBs may be overlooked. Thus the prevalence is likely underestimated.

References

1. Juilliere Y, 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: 663–5.
2. Bauters C, Chmait A, Tricot O, Lamblin N, Van Belle E, Lablanche JM. Coronary thrombosis and myocardial bridging. Circulation 2002; 105: 130.
3. 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.
4. Angelini P, Trivellato M, Donis J, Leachman RD. Myocardial bridges: a review. Prog Cardiovascular Dis 1983; 26: 75–88.
5. Hunink MG, Goldman L, Tosteson AN, Mittleman MA, Goldman PA, Williams LW, et al. The recent decline in mortality from coronary heart disease, 1980–1990; The effect of secular trends in risk factors and treatment. JAMA 1997; 277: 535–42.

6. McGovern PG, Pankow JS, Shahar E, Doliszny KM, Folsom AR, Blackburn H, et al. Recent trends in acute coronary heart disease–mortality, morbidity, medical care and risk factors. The Minnesota Heart Survey Investigators. N Engl J Med 1996; 334: 884–90.
7. Lloyd-Jones DM, Larson MG, Beiser A, Levy D. Lifetime risk of developing coronary heart disease. Lancet 1999; 353: 89–92.
8. Braunwald E. Unstable angina. A classification. Circulation 1989; 80: 410–4.
9. Geirenger E. The mural coronary. Am Heart J 1951; 41: 359–68.
10. Çay S, Ozturk S, Cihan G, Kisacik HL, Korkmaz S. Angiographic prevalence of myocardial bridging. Anadolu Kardiyol Derg 2006; 6: 9-12.
11. Ishimori T, Raizner AE, Chahine RA, Awdeh M, Luchi RJ. Myocardial bridges in man: clinical correlations and angiographic accentuation with nitroglycerin. Cathet Cardiovasc Diagn 1977; 3: 59–65.
12. 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.
13. 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.
14. Achrafi H. Hypertrophic cardiomyopathy and myocardial bridging. Int J Cardiol 1992; 37: 111–2.
15. Tauth J, Sullebarger T. Myocardial infarction associated with myocardial bridging: case history and review of the literature. Cathet Cardiovasc Diagn 1997; 40: 364–7.
16. 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.
17. 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: 965–9.
18. Cheng TO. Myocardial bridging in a young patient with sudden death. Clin Cardiol 1997; 20: 743.
19. Kulan K, Kulan C, Tuncer C, Komsuoglu B, Telatar M. Myocardial perfusion scintigraphy in a myocardial bridging of coronary artery. Clin Nucl Med 1996; 21: 888–9.
20. Ge J, Erbel R, Gorge G, Haude M, Meyer J. High wall shear stress proximal to myocardial bridging and atherosclerosis: intracoronary ultrasound and pressure measurements. Br Heart J 1995; 73: 462–5.
21. Ishii T, Asuwa N, Masuda S, Ishikawa Y. The effects of a myocardial bridge on coronary atherosclerosis and ischemia. J Pathol 1998; 185: 4–9.
22. Zoghi M, Duygu H, Nalbantgil S, Kirilmaz B, Turk U, Ozerkan F, et al. Impaired endothelial function in patients with myocardial bridge. Echocardiography 2006; 23: 577-81.
23. Ishikawa Y, Akasaka Y, Ito K, Akishima Y, Kimura M, Kiguchi H, et al. Significance of anatomical properties of myocardial bridge on atherosclerosis evolution in the left anterior descending coronary artery. Atherosclerosis 2006; 186: 380-9.
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. Klues HG, Schwarz ER, vom Dahl J, Reffelmann T, Reul H, Potthast K, et al. Disturbed intracoronary hemodynamics in myocardial bridging: early normalization by intracoronary stent placement. Circulation 1997; 96: 2905–13.
26. 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: 403–8.