Article

A Comprehensive Single-Center Retrospective Analysis of the Prevalence and Demographic Features of Myocardial Bridges in Northern Cyprus

Musa Muhtaroglu^{1,*}, Cenk Conkbayir^{2,3}

Submitted: 27 January 2025 Revised: 30 March 2025 Accepted: 22 April 2025 Published: 26 May 2025

Abstract

Background: Myocardial bridging (MB) is an anatomical variant in which segments of epicardial coronary arteries course partially within the myocardium. The prevalence of MB is reportedly as high as 15–85% in autopsy studies, whereas coronary angiography studies show a prevalence ranging from 0.5–3%. The aim of this study was to investigate the prevalence and demographic characteristics of MB in patients undergoing coronary angiography in Northern Cyprus. Methods: This retrospective review analyzed coronary angiography data for 6980 consecutive adult patients undergoing coronary angiography between December 2020 and January 2023. Results: MB was detected in 89 of 6980 patients who underwent coronary angiography, giving an angiographic prevalence of 1.27%. The mean age of patients was 59.4 ± 13.5 years (range: 26–90 years), with 28 (31.5%) females and 61 (68.5%) males. MB was most commonly observed in the left anterior descending (LAD) artery. MB was found in the middle LAD segment in 65 patients (73.0% of all cases), in the distal LAD segment in 22 patients (24.7%), and in the right coronary artery (RCA) in two patients (2.2%). Conclusion: This study provides new data on the prevalence of MB in Northern Cyprus and on regional differences in this condition. Further epidemiological research is needed to better characterize MB distribution and its potential clinical significance.

Keywords

myocardial bridging; coronary angiography; coronary artery disease

Introduction

Myocardial bridging (MB) is an anatomical variant in which segments of epicardial coronary arteries partially course within the myocardium. MB is characterized by obstruction of the overlying muscle fibers of the coronary artery during systole [1]. It is mostly observed in the middle segment of the left anterior descending (LAD) artery, but other branches may also be affected, including marginal branches, diagonal branches, and the posterior descending branch of the right coronary artery (RCA) [1,2]. This anatomical variant was first described by Reyman in 1737 with autopsy findings, and later proven by Portman and Irving in 1960 with the use of angiography [2,3]. However, the prevalence of MB in the population is unclear, and the reported prevalence in different studies is highly variable [4–6]. The overall prevalence reported in coronary angiographic series is 0.5-3%, while in autopsy series it is 15-85% [6,7]. This wide variation is due to the diversity in the definitions and assessment methods used in studies to date [6–8]. The degree of coronary stenosis caused by MB varies depending on the regional distribution of the variant, its thickness, length and the level of contraction of the heart [9,10]. MB is usually benign and mostly asymptomatic in its initial stages, with later complications ranging from myocardial ischemia to myocardial infarction, arrhythmias, heart blocks, ventricular dysfunction and sudden cardiac death [11–13]. The aim of this retrospective, single-center study was to determine the prevalence and demographic characteristics of MB in patients undergoing coronary angiography in Northern Cyprus.

Materials and Methods

Study Design and Setting

Data were collected by retrospective review of angiographic data for 6980 consecutive adult patients who underwent femoral artery coronary angiography in the Cardiology service at Dr. Burhan Nalbantoğlu State Hospital between December 2020 and January 2023. A total of 89 patients diagnosed with MB during coronary angiography were included in this study. Although some patients also presented with concomitant coronary artery disease (CAD), a distinct statistical comparison between MB patients with

¹Department of Anatomy, Faculty of Medicine, European University of Lefke, 99728 Lefke, Northern Cyprus, TR-10 Mersin, Turkey

²Department of Cardiology, Faculty of Medicine, Near East University, 99138 Nicosia, Cyprus

³Department of Cardiology, Faculty of Medicine, University of Kyrenia, 99320 Kyrenia, Cyprus

^{*}Correspondence: musamuhtaroglu@hotmail.com (Musa Muhtaroglu)

and without CAD was not conducted, as the main aim was to assess the anatomical and demographic features of MB specifically. The study protocol was approved by the Institutional Ethics Committee of Dr. Burhan Nalbantoğlu State Hospital (Approval Number: YTK.1.01 [EK 56/20]). All procedures were carried out in accordance with the principles of the Declaration of Helsinki.

Patient Selection

The inclusion criteria were: (1) patients aged 18 years or older; (2) patients undergoing coronary angiography for suspected CAD or for another clinical reason; (3) patients having complete clinical and demographic information; and (4) patients with sufficient angiographic imaging to allow for evaluation of MB. The exclusion criteria were: (1) past percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG) surgery; (2) suboptimal coronary angiographic images that impaired the precise assessment of MB; (3) patients receiving drugs that significantly affect myocardial contractility; or (4) patients with severe left ventricular dysfunction (ejection fraction <30%).

Coronary Angiography and Assessment of Myocardial Bridging

All coronary angiograms were performed using the Siemens Artis Q device. After preparation of the right femoral artery, a 6-F femoral sheath was placed using 2% lidocaine as local anesthesia. During the operation, 3000 to 5000 units of unfractionated heparin were administered intravenously according to body weight and glomerular filtration rate. Nitroglycerin (100–200 µg) and diltiazem (5 mg) were administered according to blood pressure to produce an antispasmodic effect. No absolute contraindication to the use of diltiazem was proven. The standard Judkins technique was used to visualize the coronary arteries using a 5-F diagnostic catheter inserted via the right femoral artery. Conventional biplane cine-angiography equipment was used to obtain images of each coronary artery. Two experienced interventional cardiologists independently evaluated the myocardial bridge observed along the intramyocardial course of the coronary artery during systolic compression. To determine the degree of MB, narrowing of the systolic lumen was graded according to previously defined criteria: Grade I: <50% systolic lumen narrowing, Grade II: 50–75% systolic lumen narrowing, Grade III: >75% systolic lumen narrowing [14]. The anatomic distribution of MB was recorded. The LAD, left circumflex artery (LCx), and RCA were carefully examined to determine their location within the major coronary arteries. The LAD was divided into three separate anatomic segments (proximal, mid, distal) according to the regions affected by compression during its course in the heart. Proximal LAD was defined as the segment between the first diagonal of the LAD and the level of its separation from the main left artery.

Mid-LAD was defined as the segment between the first diagonal and the second diagonal, while distal LAD was defined as the segment distal to the second diagonal.

Demographic and Clinical Data

Demographic data and laboratory results were obtained retrospectively from hospital archives. The age and gender of patients were recorded, and cardiovascular risk factors such as hypertension, diabetes mellitus, dyslipidemia and smoking status were assessed. The presence of CAD was determined by angiographic evaluation, and concomitant coronary pathologies were also documented. Biochemical parameters were obtained from hospital records, including fasting blood glucose, total cholesterol, lowdensity lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides, creatinine and hemoglobin levels. Subgroup analyses were performed according to the degree of systolic lumen compression due to MB, and potential relationships between biochemical parameters and cardiovascular risk factors were investigated.

Statistical Analysis

Statistical analyses were performed using SPSS statistical software version 16.0 (IBM Corp., Armonk, NY, USA). Data were presented as the mean and standard deviation for continuous variables, and as percentage distribution for categorical variables. Normality of data distribution was assessed using the Shapiro–Wilk test and supported by visual inspection of Q–Q plots. Baseline differences between groups were evaluated by *t*-test for continuous variables, and chi-square test for categorical variables. *p*-values < 0.05 were considered statistically significant.

Results

Of the 6980 patients who underwent coronary angiography, 89 were found to have MB, giving an angiographic prevalence of 1.27%. The age of the MB study population ranged from 26–90 years, with a mean age of 59.4 \pm 13.5 years (Table 1). Of the 89 patients with MB, 68.5% were men and 31.5% were women. Females with MB were significantly older (66.5 \pm 14.2 years) than male patients $(57.6 \pm 12.3 \text{ years})$. An independent samples t-test was conducted to compare age between the two groups. Levene's test indicated equal variances (p = 0.361), so the assumption of homogeneity was met. The difference in means was statistically significant (t (87) = -3.002, p = 0.004, 95% CI [-14.72, -2.99]). Suggesting that MB presentation may vary according to gender. Among the patients with MB, 50.6% had coronary artery disease, 37.1% had diabetes mellitus, 46.1% had hyperlipidemia, 46.1% were smokers, and 48.3% had hypertension (Table 1). The mean values for the biochemical assay results were: hemoglobin,

Table 1. Demographic Data of Patients with Myocardial Bridging.

Variable	Value			
Age (years)	59.4 ± 13.5			
Males, n (%)	61 (68.5%)			
Females, n (%)	28 (31.5%)			
Males, mean age (years)	57.6 ± 12.3			
Females, mean age (years)	66.5 ± 14.2			
Coronary artery disease, n (%)	45 (50.6%)			
Hypertension, n (%)	43 (48.3%)			
Diabetes mellitus, n (%)	33 (37.1%)			
Hyperlipidemia, n (%)	41 (46.1%)			
Glucose (mg/dL)	152 ± 59			
Urea (mg/dL)	37.1 ± 6.53			
Creatinine (mg/dL)	0.836 ± 0.146			
Hemoglobin (g/dL)	13.30 ± 1.67			
Triglycerides (mg/dL)	163 ± 46			
Total cholesterol (mg/dL)	192 ± 27.2			
HDL (mg/dL)	46.9 ± 7.83			
LDL (mg/dL)	127 ± 24.1			
Smoking status, n (%) 41 (46.1%)				
HDI high density linoprotein: I DI low				

HDL, high-density lipoprotein; LDL, low-density lipoprotein.

 13.30 ± 1.67 g/dL; creatinine, 0.836 ± 0.146 mg/dL; urea, 37.1 ± 6.53 mg/dL; and fasting blood sugar, 152 ± 59 mg/dL. The mean lipid profile results were: triglycerides, $163 \pm 46 \text{ mg/dL}$; total cholesterol, $192 \pm 27.2 \text{ mg/dL}$; HDL, 46.9 ± 7.83 mg/dL; and LDL, 127 ± 24.1 mg/dL. MB patients were classified into three groups according to the level of coronary artery compression during the systolic phase: Grade I (n = 52), Grade II (n = 19), and Grade III (n = 18). No statistically significant differences were found between these groups in terms of glucose, urea, creatinine, hemoglobin, triglyceride, total cholesterol, HDL, LDL, age, gender, hypertension, diabetes mellitus, hyperlipidemia, and smoking (Table 2). Although the Grade III group had a greater incidence of hypertension (72.2%) than the other two groups, this difference failed to reach statistical significance (p = 0.076). With regard to anatomical distribution, most MB was found in the mid-LAD (n = 65, 73.0%), followed by the distal-LAD (n = 22, 24.7%) and the RCA (n = 2, 2.2%) (Table 3). No evidence of MB was found in the LCx.

Discussion

MBs are congenital anatomical variations of the coronary arteries. Very few are symptomatic, with most usually detected as incidental findings during coronary angiography or autopsy [3,4]. Literature reviews show marked differences between autopsy and angiography studies. Autopsy studies have reported a 15–85% prevalence of MB [1,14]. One autopsy study reported a prevalence of 62% in

150 cases (104 males and 46 females) [15], while another study of 90 autopsy cases (56 males and 34 females) found a prevalence of 55.6% [16]. A large angiography study conducted in the Republic of China reported 1002 cases of MB amongst 37,105 patients, giving an overall prevalence of 2.70% [17]. A comprehensive retrospective angiographic study of 25,982 patients from Turkey found the prevalence of MB was 1.22% [18]. A separate retrospective angiographic study of 7200 patients from Turkey found 29 patients with MB, giving a prevalence of just 0.4% [19]. In the present angiographic study, MB was detected in 89 patients from a total of 6980, giving a prevalence of 1.27%. Moreover, the prevalence was significantly higher in males, with 68.5% of cases found in males and 31.5% in females. Our results are similar to the prevalence rates reported in the existing literature [1,5,9,17,18]. Angiographically, the low prevalence of 1.27% may be due to the fact that small and thin MBs exert less compression on the coronary arteries. In addition, the severity of coronary obstruction caused by MBs is related to the location, thickness, length and cardiac contractility of the bridge [6,9,10,18,20]. It has been reported that LAD is the most commonly affected artery for MB, with very rare involvement of the RCA and LCx arteries [1,3,4,17,19,20]. The available literature indicates that MB is most commonly localized in the middle segment of the LAD, usually 1–10 mm deep, and typically involving a segment of 15-25 mm in length [4,17,19,20]. Cay et al. [18] reported that MB cases in the LAD artery were almost equally distributed in the middle and distal segments, with no MB observed in the proximal segment. The differences observed between various studies are probably due to the use of different definitions and evaluation methods [6– 8,21]. Coronary angiography is the optimal detection technique for MB [1,6,13]. Other diagnostic modalities used to diagnose MB include multidetector computed tomography (MDCT), electron beam computed tomography (EBCT), intravascular ultrasound (IVUS), magnetic resonance imaging (MRI), and intravascular pressure devices (FFR), providing additional information regarding the morphology and function of MB [7,8,10-12,22]. Among these, IVUS and optical coherence tomography (OCT) have emerged as particularly valuable techniques for evaluating MB depth and its potential hemodynamic significance. IVUS enables visualization of the "halo" sign, an echolucent zone around the vessel that can indicate the depth and severity of the tunneled segment [8]. Due to its superior spatial resolution, OCT allows the detailed assessment of vessel wall structures and more accurate anatomical characterization of MBs [8,22]. Studies have shown that deeper and longer MBs may cause significant systolic compression, potentially with clinical consequences such as exertional angina, myocardial ischemia, arrhythmias, and in rare cases even sudden cardiac death [3,8,20,23]. Therefore, in order to better understand their clinical relevance and guide appropriate management strategies, MBs should not only be iden-

Heart Surgery Forum E369

Table 2. Demographic and Biochemical Comparisons Between Myocardial Bridging Groups.

	Grade I $(n = 52)$	Grade II $(n = 19)$	Grade III $(n = 18)$	<i>p</i> -value
Age (years)	59.9 ± 12.9	57.3 ± 15.3	65.0 ± 12.8	0.225
Males (n)	36 (40.4%)	13 (14.6%)	12 (13.5%)	0.980
Females (n)	16 (18.0%)	6 (6.7%)	6 (6.7%)	0.980
Coronary atery dsease (n)	23 (25.8%)	11 (12.4%)	11 (12.4%)	0.360
Hypertension (n)	22 (24.7%)	8 (9.0%)	13 (14.6%)	0.076
Diabetes mellitus (n)	18 (20.2%)	10 (11.2%)	5 (5.6%)	0.250
Hyperlipidemia (n)	24 (27.0%)	10 (11.2%)	7 (7.9%)	0.704
Glucose (mg/dL)	152.85 ± 63.91	153.47 ± 49.31	148.83 ± 56.59	0.961
Urea (mg/dL)	36.52 ± 6.46	38.84 ± 7.43	37.0 ± 5.70	0.496
Creatinine (mg/dL)	0.83 ± 0.15	0.87 ± 0.15	0.83 ± 0.14	0.550
Hemoglobin (g/dL)	13.20 ± 1.64	13.99 ± 1.60	12.99 ± 1.74	0.142
Triglycerides (mg/dL)	160.28 ± 50.19	156.16 ± 40.95	177.39 ± 36.37	0.199
Total cholesterol (mg/dL)	196.44 ± 27.65	179.05 ± 26.87	193.94 ± 23.08	0.068
HDL (mg/dL)	47.39 ± 8.09	46.74 ± 7.21	45.56 ± 7.97	0.709
LDL (mg/dL)	126.64 ± 25.63	127.26 ± 21.32	128.5 ± 23.30	0.961
Smoking status (n)	27 (30.3%)	6 (6.7%)	8 (9%)	0.281

Table 3. Distribution of myocardial bridging across coronary arteries.

	Mid-LAD	Distal LAD	LCx	RCA
Grade I	40	11	-	1
Grade II	12	7	-	-
Grade III	13	4		1
Total (n)	65	22	-	2
MB Distribution (%)	73.0	24.7	-	2.2

LAD, left anterior descending artery; LCx, left circumflex artery; RCA, right coronary artery; MB Distribution, myocardial bridging Distribution.

tified by their angiographic appearance, but also evaluated according to their depth and extent [6,8,24].

The clinical significance of MB remains controversial, with many experts viewing it as a benign anatomical variant. However, some studies have linked MB to myocardial ischemia, ventricular arrhythmias, and even sudden cardiac death [20,23]. MB may also be associated with acute coronary syndromes, coronary spasms and syncope [20,25]. Several mechanisms that may lead to the development of MB-induced CAD have been proposed in the literature. For example, the systolic compression of MB could impede coronary blood flow and trigger atherosclerosis and endothelial dysfunction. These hemodynamic changes can lead to coronary spasm, plaque rupture and direct myocardial ischemia, resulting in the development of MB-induced CAD. In patients with MB, narrowing of the coronary artery during systole, and the persistence of this narrowing throughout diastole, causes the artery to be affected even during periods of maximal coronary flow. This may result in inadequate blood supply to the cardiac tissue and trigger myocardial ischemia, leading to subendocardial or transmural ischemia of the anterior wall [20,26]. In an earlier study, MB patients were divided into three groups according to the degree of systolic compression [27]. The first group was comprised of patients with 0-30% myocardial compression, the second group of patients with 31-

50% compression, and the third group of patients with 51-100% compression. Electrocardiography (ECG) and stress tests revealed no evidence of ischemia in the first group. In the second group, 25% of patients exhibited ischemic findings in their ECGs, but none showed ischemic changes in the stress tests. In the third group, 30% of patients exhibited ischemic ECG changes and 33% showed ischemic findings in stress tests. Our study also divided MB patients into three groups based on the degree of systolic compression observed during coronary angiography. The prevalence of CAD progressively increased according to the severity of MB. In Grade I, 44.2% of patients showed CAD, increasing to 57.9% in Grade II and 61.1% in Grade III. However, despite the increasing trend, the difference between groups was not statistically significant (p = 0.36). These results are in line with previous studies that suggest a potential association between MB and CAD, but do not establish a definitive causal relationship. The trend of increasing CAD prevalence with increasing MB contractility is consistent with the hemodynamic and pathophysiological mechanisms reported in the literature. However, the fact that statistical significance was not reached suggests the association may be multifactorial, and that mechanical compression alone is not the determining factor [6,28].

Patients with anginal symptoms often require pharmacologic treatment. Beta-blockers are recommended as

first-line therapy because they can improve hemodynamic disturbances by decreasing heart rate and contractility, and increasing coronary blood flow [4,25,29]. In addition, nondihydropyridine calcium channel blockers can alleviate symptoms through their vasodilatory effects, and through this mechanism they may also be effective at modifying cardiovascular risk factors [4,29]. If atherosclerosis is detected, antiplatelet therapy is recommended to manage the patient's cardiovascular risks [30]. Surgical intervention may also be preferred to ensure the arteries with MB remain patent, with the aim being to achieve long-term symptomatic improvement and a favorable prognosis. However, the surgical option is usually limited to patients who do not respond adequately to medical therapy, as stent placement carries the risk of complications such as restenosis, stent thrombosis and perforation [4,29,30]. In one study, the rate of in-stent stenosis detected within the first 7 weeks after surgery was reported to be 36%. This highlights the frequency of vascular narrowing in stented areas during the early postoperative period, and the clinical significance of such complications. The study also provided important data on the efficacy of such interventions in the early period, and emphasizes the need to monitor patients for restenosis and other vascular complications [31]. Supra-arterial myotomy (surgical unroofing) should be considered an option for patients with hemodynamically significant MB when pharmacologic therapy is inadequate. Studies have shown this surgical intervention offers an effective alternative, especially in patients who do not respond to medical therapy and have difficulty controlling symptoms. In the pediatric population, supra-arterial myotomy can provide permanent symptom relief and eliminate the need for long-term chronic medical treatment. Therefore, surgical unroofing should be considered as a feasible option for the treatment of appropriate patients [20,32]. This surgical technique has been demonstrated to improve symptoms, reverse local myocardial ischemia, and increase coronary blood flow. Furthermore, following surgical intervention in patients with MB, coronary arteries have been observed to regain their normal function and the blood supply to cardiac tissue is improved [20,30].

Successful cases of endoscopic surgical unroofing have shown that it offers some advantages due to its minimally invasive features. However, for deeply located coronary arteries, the possibility of inadequate decompression may limit the clinical efficacy of this method. Alternative treatment options such as CABG are recommended for such cases, especially for MB that exceeds 25 mm and arterial segments that cannot be fully decompressed during diastole [20,25]. However, CABG has been reported to have limited success in some cases and is also associated with high graft failure rates. These limitations may adversely affect the efficacy of the graft, especially due to the large size of MB or to anatomical features of the arterial segments. Hence, the limited efficacy of CABG highlights the need to consider

other treatment options for certain patient groups [20,31]. In the current study, patients with symptomatic MB were treated with medical therapy, and lifestyle changes were subsequently recommended in light of the outcome. The aim was to manage cardiovascular risk factors and thus have a positive effect on overall cardiac health. Furthermore, the effectiveness of treatment can be improved by increasing physical activity levels, adopting healthy eating habits, and avoiding harmful habits such as smoking, thereby supporting the clinical recovery of patients with MB.

Limitations

This study has several limitations. First, it was a single-center retrospective analysis, which may limit the generalizability of our findings. Retrospective studies are by their nature susceptible to selection bias, and a prospective, multicenter study design would provide more robust results. Second, coronary angiography was the primary imaging modality used to detect MBs. While conventional angiography is widely used in clinical settings, it has limited ability to evaluate the depth, thickness, and intramyocardial course of MBs. Advanced imaging techniques such as IVUS, OCT, and computed tomography (CT) angiography allow for a more detailed morphological assessment and may better characterize the hemodynamic relevance of MBs. The absence of such high-resolution modalities in this study restricted our ability to quantify MB anatomy more precisely and to assess the extent of dynamic coronary compression or myocardial ischemia with greater accuracy [8,22,33]. Third, our study did not include a classification of MBs based on their anatomical depth. Increasing evidence indicates that compared with superficial bridges, deeper MBs are associated with more pronounced systolic compression and a higher likelihood of adverse clinical outcomes, including myocardial ischemia and arrhythmias [3,8,20,23]. The lack of depth-based classification prevented us from evaluating potential differences in the physiological and clinical impact of the different MB subtypes, thus potentially influencing the interpretation of symptom burden and therapeutic planning. Fourth, no longterm follow-up data were included, which is important for understanding the natural history of MB, its progression, and long-term clinical outcomes. Further studies with a longer follow-up period will be required to evaluate the prognosis of patients with MB-related systolic compression (>75%) and/or concurrent CAD [20,25]. Finally, although some patients in our study had concomitant CAD, we did not perform separate statistical comparisons between MB patient groups with and without CAD. Earlier studies suggested that MB may contribute to the development of atherosclerosis, particularly in the proximal segments, due to changes in shear stress and endothelial dysfunction [8,20,33,34]. However, this was beyond the main scope of our study.

Heart Surgery Forum E371

Conclusion

This study retrospectively evaluated the prevalence and demographic characteristics of MB in Northern Cyprus, providing new data on its regional distribution. Our results suggest a potential association between MB severity and the prevalence of CAD, however, this relationship did not reach statistical significance. Furthermore, the current findings indicate that it may be insufficient to focus solely on the narrowing of the systolic lumen in the clinical management of MB and highlights the need for multifactorial assessments. Our study also highlights the need for further research into the physiological and clinical implications of MB. In particular, prospective, multicenter, and long-term studies are warranted to better elucidate the relationship between MB and CAD, and to optimize patient-centered management strategies.

Availability of Data and Materials

The data are available from the corresponding author upon reasonable request.

Author Contributions

MM designed the research study, conducted the literature review, collected and interpreted the data, performed the statistical analysis, and drafted the manuscript. CC contributed to the interpretation of the data and critically revised the manuscript. Both authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work. Both authors contributed to editorial changes in the manuscript. Both authors read and approved the final manuscript.

Ethics Approval and Consent to Participate

The study was carried out in accordance with the guidelines of the Declaration of Helsinki and approved by the Ethics Committee of Dr. Burhan Nalbantoğlu State Hospital (Protocol No. YTK.1.01(EK 56/20)). This study was exempted from informed consent by the Ethics Committee of the Dr. Burhan Nalbantoğlu State Hospital Review Board because it was a retrospective study. The research was conducted at Dr. Burhan Nalbantoğlu State Hospital, where all clinical data were collected.

Acknowledgment

Not applicable.

Funding

This research received no external funding.

Conflict of Interest

The authors declare no conflict of interest.

References

- [1] Şenöz O, Yapan Emren Z. Is myocardial bridge more frequently detected on radial access coronary angiography? BMC Cardiovascular Disorders. 2021; 21: 564. https://doi.org/10.1186/ s12872-021-02382-y.
- [2] Tiryakioğlu M, Aliyu MN. Myocardial bridge. Folia Morphologica. 2020; 79: 411–414. https://doi.org/10.5603/FM.a2019. 0080
- [3] Matta A, Roncalli J, Carrié D. Update review on myocardial bridging: New insights. Trends in Cardiovascular Medicine. 2024; 34: 10–15. https://doi.org/10.1016/j.tcm.2022.06.002.
- [4] Lee MS, Chen CH. Myocardial Bridging: An Up-to-Date Review. The Journal of Invasive Cardiology. 2015; 27: 521–528.
- [5] Podolec J, Wiewiórka Ł, Siudak Z, Malinowski K, Dudek D, Gackowski A, et al. Prevalence and clinical presentation of myocardial bridge on the basis of the National Polish Percutaneous Interventions Registry and the Classification of Rare Cardiovascular Diseases. Kardiologia Polska. 2018; 77: 465–470. https://doi.org/10.5603/KP.a2019.0041.
- [6] Alegria JR, Herrmann J, Holmes DR, Jr, Lerman A, Rihal CS. Myocardial bridging. European Heart Journal. 2005; 26: 1159–1168. https://doi.org/10.1093/eurheartj/ehi203.
- [7] Rovera C, Moretti C, Bisanti F, De Zan G, Guglielmo M. Myocardial Bridging: Review on the Role of Coronary Computed Tomography Angiography. Journal of Clinical Medicine. 2023; 12: 5949. https://doi.org/10.3390/jcm12185949.
- [8] Sternheim D, Power DA, Samtani R, Kini A, Fuster V, Sharma S. Myocardial Bridging: Diagnosis, Functional Assessment, and Management: JACC State-of-the-Art Review. Journal of the American College of Cardiology. 2021; 78: 2196–2212. https://doi.org/10.1016/j.jacc.2021.09.859.
- [9] Soran O, Pamir G, Erol C, Kocakavak C, Sabah I. The incidence and significance of myocardial bridge in a prospectively defined population of patients undergoing coronary angiography for chest pain. The Tokai Journal of Experimental and Clinical Medicine. 2000; 25: 57–60.
- [10] Wirianta J, Mouden M, Ottervanger JP, Timmer JR, Juwana YB, de Boer MJ, et al. Prevalence and predictors of bridging of coronary arteries in a large Indonesian population, as detected by 64-slice computed tomography scan. Netherlands Heart Journal: Monthly Journal of the Netherlands Society of Cardiology and the Netherlands Heart Foundation. 2012; 20: 396–401. https://doi.org/10.1007/s12471-012-0296-4.
- [11] Angelini P, Trivellato M, Donis J, Leachman RD. Myocardial bridges: a review. Progress in Cardiovascular Diseases. 1983; 26: 75–88. https://doi.org/10.1016/0033-0620(83)90019-1.
- [12] Rajendran R, Hegde M. The prevalence of myocardial bridging on multidetector computed tomography and its relation to coronary plaques. Polish Journal of Radiology. 2019; 84: e478–e483. https://doi.org/10.5114/pjr.2019.90370.
- [13] Karna SK, Chourasiya M, Parikh RP, Chaudhari T, Patel U. Prevalence of myocardial bridge in angiographic population-A study from rural part of western India. Journal of Family

- Medicine and Primary Care. 2020; 9: 1963–1966. https://doi.org/10.4103/jfmpc.jfmpc 1075 19.
- [14] Noble J, Bourassa MG, Petitclerc R, Dyrda I. Myocardial bridging and milking effect of the left anterior descending coronary artery: normal variant or obstruction? The American Journal of Cardiology. 1976; 37: 993–999. https://doi.org/10.1016/0002-9149(76)90414-8.
- [15] Akishima-Fukasawa Y, Ishikawa Y, Mikami T, Akasaka Y, Ishii T. Settlement of Stenotic Site and Enhancement of Risk Factor Load for Atherosclerosis in Left Anterior Descending Coronary Artery by Myocardial Bridge. Arteriosclerosis, Thrombosis, and Vascular Biology. 2018; 38: 1407–1414. https://doi.org/10.1161/ATVBAHA.118.310933.
- [16] Ferreira AG, Jr, Trotter SE, König B, Jr, Décourt LV, Fox K, Olsen EG. Myocardial bridges: morphological and functional aspects. British Heart Journal. 1991; 66: 364–367. https://doi.or g/10.1136/hrt.66.5.364.
- [17] Li JJ, Shang ZL, Yao M, Li J, Yang YJ, Chen JL, et al. Angiographic prevalence of myocardial bridging in a defined very large number of Chinese patients with chest pain. Chinese Medical Journal. 2008; 121: 405–408.
- [18] Cay S, Oztürk S, Cihan G, Kisacik HL, Korkmaz S. Angio-graphic prevalence of myocardial bridging. Anadolu Kardiyoloji Dergisi: AKD = the Anatolian Journal of Cardiology. 2006; 6: 9–12.
- [19] 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 angiography. Annals of the Academy of Medicine, Singapore. 2008; 37: 49–53.
- [20] Roberts W, Charles SM, Ang C, Holda MK, Walocha J, Lachman N, et al. Myocardial bridges: A meta-analysis. Clinical Anatomy (New York, N.Y.). 2021; 34: 685–709. https://doi.org/10.1002/ca.23697.
- [21] Murtaza G, Mukherjee D, Gharacholou SM, Nanjundappa A, Lavie CJ, Khan AA, et al. An Updated Review on Myocardial Bridging. Cardiovascular Revascularization Medicine: Including Molecular Interventions. 2020; 21: 1169–1179. https://doi.org/10.1016/j.carrev.2020.02.014.
- [22] 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. Catheterization and Cardiovascular Interventions: Official Journal of the Society for Cardiac Angiography & Interventions. 2002; 56: 58–63. https://doi.org/10. 1002/ccd.10151.
- [23] Monroy-Gonzalez AG, Alexanderson-Rosas E, Prakken NHJ, Juarez-Orozco LE, Walls-Laguarda L, Berrios-Barcenas EA, et al. Myocardial bridging of the left anterior descending coronary artery is associated with reduced myocardial perfusion reserve: a ¹³N-ammonia PET study. The International Journal of Cardiovascular Imaging. 2019; 35: 375–382. https://doi.org/10.1007/s10554-018-1460-8.
- [24] Santucci A, Jacoangeli F, Cavallini S, d'Ammando M, de An-

- gelis F, Cavallini C. The myocardial bridge: incidence, diagnosis, and prognosis of a pathology of uncertain clinical significance. European Heart Journal Supplements: Journal of the European Society of Cardiology. 2022; 24: I61–I67. https://doi.org/10.1093/eurheartjsupp/suac075.
- [25] Yuan SM. Myocardial Bridging. Brazilian Journal of Cardiovascular Surgery. 2016; 31: 60–62. https://doi.org/10.5935/ 1678-9741.20150082.
- [26] Teragawa H, Oshita C, Ueda T. The Myocardial Bridge: Potential Influences on the Coronary Artery Vasculature. Clinical Medicine Insights. Cardiology. 2019; 13: 1179546819846493. https://doi.org/10.1177/1179546819846493.
- [27] 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. American Heart Journal. 1982; 103: 283–288. https://doi.org/10.1016/ 0002-8703(82)90500-2.
- [28] Tarantini G, Barioli A, Nai Fovino L, Fraccaro C, Masiero G, Iliceto S, et al. Unmasking Myocardial Bridge-Related Ischemia by Intracoronary Functional Evaluation. Circulation. Cardiovascular Interventions. 2018; 11: e006247. https://doi.org/10.1161/CIRCINTERVENTIONS.117.006247.
- [29] Hongo Y, Tada H, Ito K, Yasumura Y, Miyatake K, Yamagishi M. Augmentation of vessel squeezing at coronary-myocardial bridge by nitroglycerin: study by quantitative coronary angiography and intravascular ultrasound. American Heart Journal. 1999; 138: 345–350. https://doi.org/10.1016/s0002-8703(99) 70123-7.
- [30] Mirzai S, Patel B, Balkhy HH. Robotic totally endoscopic off-pump unroofing of left anterior descending coronary artery myocardial bridge: A report of two cases. Journal of Cardiac Surgery. 2019; 34: 735–737. https://doi.org/10.1111/jocs.14094.
- [31] 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 (British Cardiac Society). 2000; 84: 403–408. https://doi.org/10.1136/heart.84.4.403.
- [32] Hillman ND, Mavroudis C, Backer CL, Duffy CE. Supraarterial decompression myotomy for myocardial bridging in a child. The Annals of Thoracic Surgery. 1999; 68: 244–246. https://doi.org/ 10.1016/s0003-4975(99)00482-8.
- [33] Corban MT, Hung OY, Eshtehardi P, Rasoul-Arzrumly E, Mc-Daniel M, Mekonnen G, *et al.* Myocardial bridging: contemporary understanding of pathophysiology with implications for diagnostic and therapeutic strategies. Journal of the American College of Cardiology. 2014; 63: 2346–2355. https://doi.org/10.1016/j.jacc.2014.01.049.
- [34] Ishikawa Y, Akasaka Y, Akishima-Fukasawa Y, Iuchi A, Suzuki K, Uno M, et al. Histopathologic profiles of coronary atherosclerosis by myocardial bridge underlying myocardial infarction. Atherosclerosis. 2013; 226: 118–123. https://doi.org/10.1016/j.atherosclerosis.2012.10.037.

Heart Surgery Forum E373