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MYOCARDIAL BRIDGE: A CLINICAL REVIEW OF DIAGNOSTIC, PROGNOSTIC AND THERAPEUTIC IMPLICATIONS

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ABSTRACT
Myocardial bridge (MB) is a congenital coronary anomaly, in which a segment of coronary artery travels into the myocardium instead of the normal epicardial course. Although MB can be found in any coronary artery, the most common site of myocardial bridging is in the mid left anterior descending (LAD) coronary artery. The involvement of other coronary arteries, such as the right coronary artery (RCA), diagonal, ramus, and the marginal branch, are less common. The characteristic feature of MB is that with each systole, the coronary artery is compressed. In the vast majority of cases, myocardial bridge remains clinically silent, but sometimes the patient with myocardial bridge may present with symptoms, such as exertional chest pain, myocardial infarction, arrhythmias and even sudden death. The first-line therapy of symptomatic bridging remains medical treatment with beta blockers and calcium-channel blockers, but nitrates are contraindicated due to secondary tachycardia and hypercontractility. Patients who respond poorly to the medical treatment, warrant a surgical intervention. Surgical intervention includes myotomy, intracoronary stent and coronary artery bypass grafting surgery (CABG). For the symptomatic patients, myotomy is preferred surgical procedure. Use of coronary stents is limited due to complications and unsatisfactory long-term results.

KEYWORDS: Myocardial bridge, coronary artery, myocardial infarction, arrhythmias, myotomy.

INTRODUCTION

MB is a congenital anomaly, in which a segment of a major coronary artery is surrounded by the myocardium. The coronary vessels, normally are located in the epicardium, but when a portion of the epicardial coronary artery dip down and goes intramurally through the myocardium beneath the muscle fibers for varying length & depth and then reappears again on the hearts surface, after its tunneling the artery is called “tunneled artery” and the cardiac muscle over the tunnel is called “myocardial bridge”. MB is most commonly found in the LAD coronary artery.[1-3] This condition was first identified by Rayman,[4] Portman & Ingrid were the first to identify MB radiologically,[5] Gringer et al were the first to conduct in-depth analysis by autopsy.[6] MB is characterized by” milking effect”, which is compression and narrowing of a tunneled coronary artery by the overlying myocardial tissue during systole and widening during diastole.[2,7-9] There is a close relationship between the degree of systolic narrowing of the MB and the clinical symptoms of patients.[10] MB is relatively silent in most of the cases, but sometimes it may cause symptoms such as myocardial infarction, syncope, atrioventricular block, ventricular tachycardia and sudden death.[11-14]

PREVALENCE

It is understood that myocardial bridges are common, however, in most patients, myocardial bridging is an incidental finding. It remains underdiagnosed due to the fact that only a minority of patient’s present symptoms as well as the lack of availability and, consequently, the restricted use of more accurate diagnostic methods. There is a wide discrepancy in the reported prevalence of myocardial bridging, this large variation may be explained by the use of different mode of evaluating techniques (e.g. computed tomography angiography (CTA), intravascular ultrasound (IVUS), conventional angiography or autopsy), and recent improvements in computed tomography (CT) technology which provide high spatial resolution and helps in more precise analysis. In addition, which vessels are examined, and which definition of a bridge is applied (e.g. only a “deep” bridge vs both “superficial” and “deep” bridges also the length of the bridged artery). Perhaps the most fundamental variable is whether an MB is even considered.[15-17]

Although the prevalence rate of myocardial bridging is between 15% and 85% in autopsy studies, it is only seen in 0.5–2.5% of angiographic studies.[3,18] On the other hand, the rate rises to 40% with the provocation test used

during conventional angiography.[2] The reported prevalence of the intramural course of the coronary arteries on coronary CTA finds bridged coronary segments at rates similar to the autopsy series which ranges from 6% to 58%.[15,19,20] And also, the presence of atherosclerotic plaques proximal to myocardial bridging may cause underdiagnoses.[2]

Although MB can be found in any coronary artery, the most common site of myocardial bridging is in the mid left anterior descending (LAD) coronary artery. The involvement of other coronary arteries, such as right coronary artery (RCA), diagonal, ramus, and the marginal branch, are less common,[21,22] and the main angiographic finding is systolic compression of the involved epicardial coronary artery.[5] Quantitative coronary angiography,[10] intracoronary Doppler studies, and intravascular Ultrasonography[10,23] have documented a characteristic diastolic flow disturbance. The degree of coronary obstruction by the myocardial bridge depends on factors such as location, thickness, length of the muscle bridge, and degree of cardiac contractility.

PATHOPHYSIOLOGY

Myocardial bridge and its physiology are not sufficiently understood. MB are usually small and do not have any clinically significant role, but several autopsies and intravascular ultrasound studies have shown that the segment proximal to the region of MB has been prone to developing atherosclerosis., while the intramural and segment distal to bridged artery remain free from atherosclerotic disease.[8,24,25] Both hemodynamic and structural changes play an important role in the tropism and the development of atherosclerotic plaques in coronary arteries. The role of fluid mechanics, at the entrance of MB, is important in plaque formation because disturbed near-wall blood flow patterns are a central factor in the spatial distribution of atherosclerosis,[26,27] low and oscillatory wall shear stress (WSS) are associated with increased expression of vascular cell adhesion molecule,[26,28] and reactive oxygen species production[29] as well as the development of proatherogenic endothelial cell phenotype.[27] Several autopsy studies have shown that coronary segments immediately proximal to the intramural artery, where WSS is low, have structurally dysfunctional, flat and polygonal endothelial cells, whereas endothelial lining bridged segments, where WSS is physiological or high, are structurally intact.[30] Numerous factors such as abnormal flow, osteal, and bifurcated and branching segments of arteries may induce WSS, whereas laminar flow property is less likely to be associated with AP.[31] Heart rate, blood pressure, thickness and diameter of the arterial wall, hypercholesterolemia and hyperglycemia are among the additional factors which have a significant role in the localization and progression of AP.[32,33]

Within the intramural coronary artery, increased mechanical loads likely contribute to constrictive vascular remodeling as an attempt to restore loads to homeostatic levels.[34] In patients with left ventricular hypertrophy, these mechanisms are amplified with diastolic dysfunction. In addition, the protective mechanism against the development of atherosclerosis may be related to the separation of the bridged segment from perivascular adipose tissue in the epicardium that is associated with proinflammatory cytokines and adipokines.[35] These factors likely contribute to plaque formation proximal to myocardial bridges and exert an atheroprotective role within the bridge. The segment distal to the MB are relatively free from atherosclerosis despite the presence of low WSS is not well understood.

CLINICAL MANIFESTATION

Traditionally, among the general population myocardial bridge is a relatively common and was considered usually a benign pathology, affecting mainly patients at low risk for coronary artery disease. In patients with myocardial bridging, symptoms often manifest during exercise and with tachycardia.[36] The patient may manifest chest squeezing at rest.[37] In young patients with myocardial bridging, they may have an acute anterior myocardial infarction due to a subtotal occlusion of the mid-left anterior descending coronary artery caused by myocardial bridging.[38] Symptomatic patients with myocardial bridging may present with ischemia and acute coronary syndromes,[7,11,38] coronary spasm,[12,22] ventricular septal rupture,[39] arrhythmias (including supraventricular tachycardia and ventricular tachycardia),[13] exercise-induced atrioventricular conduction blocks,[40] transient ventricular dysfunction[41] and sudden death.[42] The diagnosis of clinically important myocardial bridging should be considered in patients who have angina and do not have the traditional risk factors and the evidence of ischemia.[43,44] However, objective signs of ischemia cannot always be demonstrated in patients with myocardial bridging, most likely because of a large variability.[45] From recent studies we can say the prognosis of patients with myocardial bridges is not benign as it was believed to be in the past. The clinical manifestation of the patients with myocardial bridging can appear in two ways: firstly, by the contraction of myocardial bridge fibers and direct compression of the tunneled segment, and secondly by stimulation and acceleration of atherosclerosis in the segment proximal to the myocardial bridging.[46] The first mechanism in the young people leads mainly to coronary insufficiency, particularly in those exposed to the psychophysical exertion, while in elderly people consequences of the latter mechanism appear most frequently.[47] When myocardial bridging is associated with heart valve disorder or cardiomyopathies, the patient's symptoms can be different. Sustained elevated troponin level suggests the presence of myocardial ischemia.[48]

DIAGNOSTIC TECHNIQUES

The diagnosis of MB is usually established by the chance in patients who are examined by CAG or CTCA for various reasons, but not because they are suspected of

having MB. As there is the lack of a true gold standard for diagnosis and investigation of the anatomical and physiological significance of MB, a number of different diagnostic modalities have been used. There are essential differences between the diagnostic information provided by different diagnostic modalities.

Non-Invasive Diagnostic Techniques:

Non-invasive diagnostic techniques include Multiple-slice computed tomography (MSCT), stress single photon emission computed tomography (SPECT) and stress echocardiography. In recent studies, the use of CT to evaluate MB have detected intra myocardial segments at much higher rates than by angiography.[19,20,49] CTCA clearly demonstrates bridged segment and the overlying muscular bands but provides no direct information about the dynamic effect on the involved vessel. To estimate the degree of systolic narrowing by reconstruction of the image at different phases of the cardiac cycle, numerous attempts have been made.[50] The use of stress (SPECT) can detect reversible myocardial perfusion in patients with MB and helps to relate the amount of ischemia to the degree of systolic luminal narrowing.[51,52] Several studies have suggested the use of contrast stress echocardiography for the detection of MB but are less validated.[53]

Invasive Diagnostic Techniques:

It includes coronary angiography (CAG), intracoronary doppler, intravascular ultrasound. In CAG the muscular band overlying the artery is not shown, but it demonstrates primarily the effect on the artery, that is; systolic compression of the artery with narrowing of the lumen and diastolic relaxation, which is known as 'milking effect'.[5,18] The CAG allows an accurate assessment of the atrial lumen dimension throughout the cardiac cycle, provided the recording speed is not too low. It might also show a sudden deviation towards the septum and backwards during the LAD, which is suggestive of a partial intraseptal course, which is also known as a 'step down-step up' phenomenon.[54] In intracoronary doppler study the doppler-tipped guidewires helped in the accurate measurement of intracoronary flow velocity for the first time.[55] A characteristic "spike-and-dome" pattern or "fingertip tip" phenomenon is revealed after interrogation of MB, with abrupt early diastolic flow acceleration, rapid mid-diastolic flow deceleration, and a mid-to-late diastolic plateau.[56] The characteristic findings on IVUS, is the "half-moon" sign, an echo lucent area present only between the bridged coronary segment and epicardial tissue that persists throughout the cardiac cycle. It also can characterize sub-angiography atherosclerosis proximal to the bridge.[8]

PROGNOSIS

There are not many studies available on the prognostic implications of the MB, but there is generally a good long-term prognosis in patients with isolated MB. In one study the five-year survival of patients with isolated MB was the same as angiographically similar patients without MB, with neither of the 2-death mentioned there occurred due to MB.[57] In another 11-year follow-up study, which had a group of 61 patients with MB in the LAD, there were no cardiac deaths or acute myocardial infarction.[58] In a more recent four-year follow-up study of 118 patients by Cicek et al, there were no major cardiac events or need for revascularization.[59] It is reassuring to know all the facts and outcomes of these studies, but they do not prove that MB is an innocent bystander anomaly in all cases. Since the number of patients included in the follow-up studies are relatively small and follow-up periods are limited and MB has been implicated as a potential cause of sudden death in many other studies. In a study, Desseigne et al analyzed 19 cases with MB from a series of 930 medicolegal autopsy study, the author concluded that MB couldn't be ruled out as a responsible factor for sudden death.[60] Morlan et al in an autopsy study, found myocardial lesions that were indicative of ischemia in 22 out of 39 hearts with MB, among them all had deep MB and 13 people had sudden death.[14] All these studies suggest that MB may be one of the causes of sudden death, but given the very low incidence of sudden and unexpected death in young patients, including athletes.[61] From the fact that high prevalence of MB from the autopsy and CTCA studies, and the small proportion of these cases in which MB may be the responsible factor of sudden death it is justified that in patients with MB the probability of sudden death is extremely low.[62]

MANAGEMENT

The overall character of isolated MB usually shows a benign condition. In asymptomatic patients, therapeutic intervention is not warranted, but in symptomatic patient's therapy may be initiated to improve quality of life, particularly if there is objective evidence of myocardial ischemia. The main treatment strategy can be divided into three groups: medical therapy, PCI and surgical therapy. Obviously, medical therapy should be the first and principal strategy, and interventions should be limited to patients with refractory angina despite medical therapy.

Medical Therapy

Symptomatic patients with MB must be treated, and medication is considered first-line therapy. Medical therapy should primarily include β-blockers because β-blockers relieves the patient from hemodynamic impairment caused by MB, which decreases the heart rate, reduces the compression and contractility by the muscular band on the tunneled coronary artery, and increasing the diastolic coronary filling.[2,3,18] The use of calcium-channel blocker particularly calcium-channel blocker with negative chronotropic effect can be an alternative when the use of β-blockers is not well tolerated or contraindicated.[63] While the use of nitrates, might improve cardiac contractility and may relieve symptoms, it is advised that vasodilating agents including nitroglycerin should be avoided, as they

exacerbate symptoms by intensifying systolic compression of the bridged segment and vasodilating segments proximal to the bridge.[64,65] As the proximal segment of the tunneled artery is frequently found with atherosclerotic changes, administration of anti-atherosclerotic drugs such as anti-platelet drugs and statins may be considered as a preventive measure.[62,66]

PCI

PCI is the therapeutic option if the result of medical management is insufficient. Stables et al. reported the first case of coronary stenting for severe bridging refractory to medical therapy.[67] From these early studies evaluating this option have shown that stenting can resolve hemodynamic abnormalities and symptoms.[24] However, multiple studies have shown high rates of target lesion revascularization with PCI. In a study by T. Sujita K et al. involving 70 patients with MB who received stents (primarily drug-eluting stents) for a LAD artery lesion proximal to the bridge, they reported rates of target lesion revascularization at 1 year were much higher in patients whose stents extended into the bridged segment.[68] In a study by Haager PK et al, they reported that the patients with stent implantation experienced acceptable results.[69] However, multiple studies have suggested different complication of PCI such as coronary perforation,[70-72] in-stent restenosis,[68,69] stent fracture[73] and stent thrombosis.[74] Kunamneni et al in one study found that the patients with stents experienced more adverse events than patients who received solely medical therapy.[75] Therefore, the use of PCI has been limited in these conditions.

Surgery

Surgical intervention for the treatment of MB involves either supra-arterial myotomy or CABG. It still remains unclear which, among myotomy and CABG are the superior procedure. The cardiac muscle is dissected carefully and completely, in a typical myotomy case. In surgical myotomy, the symptoms are decreased and are associated with the reversal of local myocardial ischemia.[76] Stables et al suggested in isolated disease of a solitary artery MB, myotomy may be more appropriate than bypass surgery.[67] However there is some potential complication of myotomy, such as a mural aneurysm, perforation of the right ventricle, post-operative bleeding and formation of scars with ensuring recurrent coronary artery compression.[56,77,78] On the other hand, CABG is preferred in cases of extensive (>25 mm) or deep (>5 mm) myocardial bridges (the risk of myotomy can be considerable) or when the bridged coronary segment fails to decompress completely in diastole (myotomy is unlikely to correct the persistent diastolic compression).[79,80] The left internal thoracic artery has been used with some success as a graft.[78] CABG is also associated with the risk of graft closure secondary to competitive flow given that the bridged native LAD is dynamically compressed yet remains patent.[81] Moreover, given the persistent compression, the local ischemia is not addressed by CABG. As such, failure of CABG to improve symptoms has been associated with the need for subsequent unroofing.[82] Unroofing definitively corrects the anatomic defect, thereby improving flow and relieving the source of myocardial ischemia,[83] and can be performed via sternotomy on or off cardiopulmonary bypass or via a minimally invasive approach.[84-87]

CONCLUSION

The myocardial bridge is a congenital anomaly, most often it is located in the left anterior descending coronary artery. There is evidence that the arterial segment proximal to the myocardial bridge has a higher frequency of atherosclerosis, whereas the bridged portion itself is "spared" from atherosclerosis. Despite usually being a benign condition MB can occasionally generate a series of severe cardiovascular events, such as myocardial infarction, arrhythmia and sudden death. First-line therapy involves medical treatment with beta-blockers and non- dihydropyridine calcium-channel blockers, while nitrates are contraindicated due to secondary tachycardia and hypercontractility. For refractory symptoms, multiple interventional strategies have been attempted, such as coronary artery bypass, surgical myotomy, and stenting.

REFERENCES

1. A. G. Ferreira, S. E. Trotter, B. König, L. V Décourt, K. Fox, and E. G. Olsen, "Myocardial bridges: morphological and functional aspects.," *Br. Heart J.*, 1991; 66(5): 364–7.
2. S. Möhlenkamp, W. Hort, J. Ge, and R. Erbel, "Update on myocardial bridging," *Circulation*, 2002; 106(20): 2616–2622.
3. J. R. Alegria, J. Herrmann, D. R. Holmes, A. Lerman, and C. S. Rihal, "Myocardial bridging," *Eur. Heart J.*, 2005; 26(12): 1159–1168.
4. Reyman HC. Disertatio de vasis cordis propriis. Med Diss Univ Göt- tingen. 7th Sept 1737; 1–32.
5. W. PORSTMANN and J. IWIG, "[Intramural coronary vessels in the angiogram].," *Fortschr. Geb. Rontgenstr. Nuklearmed.*, Feb. 1960; 92: 129–33.
6. Geiringer E. The mural coronary. Am Heart J., 1951; 41: 359–368.
7. J. Noble, M. G. Bourassa, R. Petitclerc, and I. Dyrda, "Myocardial bridging and milking effect of the left anterior descending coronary artery: Normal variant or obstruction?," *Am. J. Cardiol.*, 1976; 37(7): 993–999.
8. J. Ge *et al.*, "New signs characteristic of myocardial bridging demonstrated by intracoronary ultrasound and Doppler.," *Eur. Heart J.*, 1999; 20(23): 1707–16.
9. A. R., G. H., and E. Y., "Myocardial bridging as a cause of acute myocardial infarction: A case report," *BMC Cardiovasc. Disord.*, p. no pagination, 2: 2002.
10. E. R. Schwarz, H. G. Klues, J. V. Vom Dahl, I. Klein, W. Krebs, and P. Hanrath, "Functional, angiographic and intracoronary Doppler flow characteristics in symptomatic patients with myocardial bridging: Effect of short-term intravenous beta-blocker medication," *J. Am. Coll.*

Cardiol., 1996; 27(7): 1637–1645.
11. L. Rossi *et al.*, "Myocardial bridges and ischemic heart disease.," *Eur. Heart J.*, 1980; 1(4): 239–45.
12. J. F. Berry, G. O. von Mering, C. Schmalfuss, J. A. Hill, and R. A. Kerensky, "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(1): 58–63.
13. H. Feld, V. Guadanino, G. Hollander, A. Greengart, E. Lichstein, and J. Shani, "Exercise-induced ventricular tachycardia in association with a myocardial bridge," *Chest*, 1991; 99(5): 1295–1296.
14. A. R. Morales, R. Romanelli, L. G. Tate, R. J. Boucek, and E. de Marchena, "Intramural left anterior descending coronary artery: Significance of the depth of the muscular tunnel," *Hum. Pathol.*, 1993; 24(7): 693–701.
15. R. Nakanishi, R. Rajani, Y. Ishikawa, T. Ishii, and D. S. Berman, "Myocardial bridging on coronary cta: An innocent bystander or a culprit in myocardial infarction?," *J. Cardiovasc. Comput. Tomogr.*, 2012; 6(1): 3–13.
16. C. Burnsides, J. C. Edwards, a I. Lansing, and R. L. Swarm, "Arteriosclerosis in the intramural and extramural portions of coronary arteries in the human heart.," *Circulation*, 1956; 13(2): 235–41.
17. P. Poláček, "Relation of myocardial bridges and loops on the coronary arteries to coronary occlusions," *Am. Heart J.*, 1961; 61(1): 44–52.
18. M. G. Bourassa, A. Butnaru, J. Lespérance, and J. C. Tardif, "Symptomatic myocardial bridges: Overview of ischemic mechanisms and current diagnostic and treatment strategies," *J. Am. Coll. Cardiol.*, 2003; 41(3): 351–359.
19. E. Konen, O. Goitein, L. Sternik, Y. Eshet, J. Shemesh, and E. Di Segni, "The Prevalence and Anatomical Patterns of Intramuscular Coronary Arteries. A Coronary Computed Tomography Angiographic Study," *J. Am. Coll. Cardiol.*, 2007; 49(5): 587–593.
20. L. La Grutta *et al.*, "Prevalenza dei decorsi miocardici e relazione con l'aterosclerosi coronarica studiate mediante angiografia coronarica con TC a 64 strati," *Radiol. Medica*, 2009; 114(7): 1024–1036.
21. R. G. Irvin, "The angiographic prevalence of myocardial bridging in man," *Chest*, 1982; 81(2): 198–202.
22. Y. Saito *et al.*, "Relation between severity of myocardial bridge and vasospasm," *Int. J. Cardiol.*, 2017; 248: 34–38.
23. J. Ge *et al.*, "Comparison of intravascular ultrasound and angiography in the assessment of myocardial bridging.," *Circulation*, 1994; 89(4): 1725–32.
24. T. Ishii, N. Asuwa, S. Masuda, and Y. Ishikawa, "The effects of a myocardial bridge on coronary atherosclerosis and ischaemia," *Journal of Pathology*, 1998; 185(1): 4–9.
25. Y. Ishikawa *et al.*, "Anatomic properties of myocardial bridge predisposing to myocardial infarction," *Circulation*, 2009; 120(5): 376–383.
26. C. Cheng *et al.*, "Atherosclerotic lesion size and vulnerability are determined by patterns of fluid shear stress," *Circulation*, 2006; 113(23): 2744–2753.
27. A. M. Malek, "Hemodynamic Shear Stress and Its Role in Atherosclerosis," *JAMA*, 1999; 282(21): 2035.
28. P. L. Walpola, A. I. Gotlieb, M. I. Cybulsky, and B. L. Langille, "Expression of ICAM-1 and VCAM-1 and Monocyte Adherence in Arteries Exposed to Altered Shear Stress," *Arterioscler. Thromb. Vasc. Biol.*, 1995; 15(1): 2–10.
29. J. S. McNally *et al.*, "Role of xanthine oxidoreductase and NAD(P) H oxidase in endothelial superoxide production in response to oscillatory shear stress," *Am J Physiol Hear. Circ Physiol*, 2003; 285(6): H2290-7.
30. T. Ishii, N. Asuwa, S. Masuda, Y. Ishikawa, H. Kiguchi, and K. Shimada, "Atherosclerosis suppression in the left anterior descending coronary artery by the presence of a myocardial bridge: an ultrastructural study.," *Mod. Pathol.*, 1991; 4(4): 424–431.
31. D. N. Ku, D. P. Giddens, C. K. Zarins, and S. Glagov, "Pulsatile flow and atherosclerosis in the human carotid bifurcation. Positive correlation between plaque location and low oscillating shear stress," *Arterioscler. Thromb. Vasc. Biol.*, 1985; 5(3): 293–302.
32. M. Aparci, C. Ozturk, S. Balta, S. Okutucu, and Z. Isilak, "Hypercholesterolemia is Accounted for Atherosclerosis at the Proximal Arterial Segments of Myocardial Bridging: A Pilot Study," *Clin. Appl. Thromb.*, 2016; 22(3): 297–302.
33. S. Glagov, C. Zarins, D. P. Giddens, and D. N. Ku, "Hemodynamics and atherosclerosis. Insights and perspectives gained from studies of human arteries," *Archives of Pathology and Laboratory Medicine*, Oct-1988; 112(10): 1018–1031.
34. J. D. Humphrey, "Vascular adaptation and mechanical homeostasis at tissue, cellular, and sub-cellular levels," *Cell Biochemistry and Biophysics*, 2008; 50(2): 53–78.
35. S. N. Verhagen *et al.*, "Relationship between myocardial bridges and reduced coronary atherosclerosis in patients with angina pectoris," *Int. J. Cardiol.*, 2013; 167(3): 883–888.
36. I. Dursun *et al.*, "Treatment strategies in myocardial bridging: a case report.," *Cardiovasc. Revasc. Med.*, Jul. 2006; 7(3): 195–8.
37. H. Teragawa, "Case of angina pectoris at rest and during effort due to coronary spasm and myocardial bridging," *World J. Cardiol.*, 2015; 7(6): 367.
38. J. Tauth and T. Sullebarger, "Myocardial infarction associated with myocardial bridging: case history and review of the literature.," *Cathet. Cardiovasc. Diagn.*, Apr. 1997; 40(4): 364–7.
39. R. A. Tio and T. Ebels, "Ventricular septal rupture caused by myocardial bridging," *Ann. Thorac. Surg.*,

2001; 72(4): 1369–1370.
40. K. Den Dulk, P. Brugada, S. Braat, B. Heddle, and H. J. J. Wellens, "Myocardial bridging as a cause of paroxysmal atrioventricular block," *J. Am. Coll. Cardiol.*, 1983; 1(3): 965–969.
41. M. Galli, A. Politi, and S. Zerboni, "'Functional myocardial bridging' and 'hyperkinetic state': a rare association as a cause of acute myocardial infarction," *G Ital Cardiol*, 1997; 27(12): 1286–1289.
42. A. R. Morales, R. Romanelli, and R. J. Boucek, "The mural left anterior descending coronary artery, strenuous exercise and sudden death," *Circulation*, 1980; 62(2): 230–237.
43. K. Kulan, C. Kulan, C. Tuncer, B. Komsuoğlu, and M. Telatar, "Myocardial perfusion scintigraphy in a myocardial bridging of coronary artery.," *Clin. Nucl. Med.*, Nov. 1996; 21(11): 888–9.
44. Y. Lee, D. Moon, J. Shin, S. Park, S. Park, and H. Lee, "Dipyridamole TI-201 SPECT imaging in patients with myocardial bridging," *Clin. Nucl. Med.*, 1999; 24(10): 759–64.
45. P. Angelini, M. Trivellato, J. Donis, and R. D. Leachman, "Myocardial bridges: a review.," *Prog. Cardiovasc. Dis.*, 26(1): 75–88.
46. A. Lujinović, A. Kulenović, E. Kapur, and R. Gojak, "Morphological aspects of myocardial bridges," *Bosn. J. Basic Med. Sci.*, 2013; 13(4): 212–217.
47. Y. Ishikawa, Y. Kawawa, E. Kohda, K. Shimada, and T. Ishii, "Significance of the Anatomical Properties of a Myocardial Bridge in Coronary Heart Disease," *Circ. J.*, 2011; 75(7): 1559–1566.
48. M. Dupont, W. Mullens, B. De Bruyne, and H. Vanermen, "Myotomy through heart-port access for myocardial bridging.," *Int. J. Cardiol.*, Feb. 2008; 124(1): e16-8.
49. P. J. Kim *et al.*, "Frequency of myocardial bridges and dynamic compression of epicardial coronary arteries:A comparison between computed tomography and invasive coronary angiography," *Circulation*, 2009; 119(10): 1408–1416.
50. R. De Rosa *et al.*, "Prevalence of coronary artery intramyocardial course in a large population of clinical patients detected by multislice computed tomography coronary angiography.," *Acta Radiol.*, 2008; 49(8): 895–901.
51. R. Gawor *et al.*, "Myocardial perfusion GSPECT imaging in patients with myocardial bridging," *J. Nucl. Cardiol.*, 2011; 18(6): 1059–1065.
52. E. M. Galbraith, P. Eshtehardi, and H. Samady, "SPECT perfusion imaging and myocardial bridges: Bridging the gap of diagnostic uncertainty," *Journal of Nuclear Cardiology*, 2011; 18(6): 1000–1002.
53. N. Gaibazzi, F. Rigo, and C. Reverberi, "Severe coronary tortuosity or myocardial bridging in patients with chest pain, normal coronary arteries, and reversible myocardial perfusion defects.," *Am. J. Cardiol.*, 2011; 108(7): 973–978.
54. S. Leschka *et al.*, "Myocardial Bridging: Depiction Rate and Morphology at CT Coronary Angiography—Comparison with Conventional Coronary Angiography," *Radiology*, 2008; 246(3): 754–762.
55. R. a Tio, I. C. Van Gelder, P. W. Boonstra, and H. J. Crijns, "Myocardial bridging in a survivor of sudden cardiac near-death: role of intracoronary doppler flow measurements and angiography during dobutamine stress in the clinical evaluation.," *Heart*, 1997; 77(3): 280–282.
56. H. G. Klues *et al.*, "Disturbed Intracoronary Hemodynamics in Myocardial Bridging," *Circulation*, 1997; 96(9): 2905 LP-2913.
57. J. R. Kramer, H. Kitazume, W. L. Proudfit, and F. M. Sones, "Clinical significance of isolated coronary bridges: Benign and frequent condition involving the left anterior descending artery," *Am. Heart J.*, 1982; 103(2): 283–288.
58. Y. Juillière, V. Berder, C. Suty-Selton, P. Buffet, N. Danchin, and F. Cherrie, "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): 663–665.
59. D. Çiçek, N. Kalay, and H. Müderrisoğlu, "Incidence, clinical characteristics, and 4-year follow-up of patients with isolated myocardial bridge: A retrospective, single-center, epidemiologic, coronary arteriographic follow-up study in southern Turkey," *Cardiovasc. Revascularization Med.*, 2011; 12(1): 25–28.
60. P. Desseigne, A. Tabib, and R. Loire, "[Myocardial bridging on the left anterior descending coronary artery and sudden death. Apropos of 19 cases with autopsy]," *Arch Mal Coeur Vaiss*, 1991; 84(4): 511–516.
61. B. J. Maron, J. J. Doerer, T. S. Haas, D. M. Tierney, and F. O. Mueller, "Sudden deaths in young competitive athletes analysis of 1866 deaths in the united states, 1980-2006," *Circulation*, 2009; 119(8): 1085–1092.
62. A. V. G. Bruschke, C. E. Veltman, M. A. de Graaf, and H. W. Vliegen, "Myocardial bridging: What have we learned in the past and will new diagnostic modalities provide new insights?," *Netherlands Hear. J.*, 2013; 21(1): 6–13.
63. O. H. Kracoff, I. Ovsyshcher, and M. Gueron, "Malignant course of a benign anomaly: Myocardial bridging," *Chest*, 1987; 92(6): 1113–1115.
64. Y. Hongo, H. Tada, K. Ito, Y. Yasumura, K. Miyatake, and M. Yamagishi, "Augmentation of vessel squeezing at coronary-myocardial bridge by nitroglycerin: Study by quantitative coronary angiography and intravascular ultrasound," *Am. Heart J.*, 1999; 138(2): 345–350.
65. V. B. Carvalho *et al.*, "Hemodynamic determinants of coronary constriction in human myocardial bridges.," *Am. Heart J.*, Jul. 1984; 108(1): 73–80.
66. S.-M. Yuan, "Myocardial bridging," *Brazilian J. Cardiovasc. Surg.*, 2015; 31(1): 2015–2017.
67. R. H. Stables, C. J. Knight, J. G. McNeill, and U.

Sigwart, "Coronary stenting in the management of myocardial ischaemia caused by muscle bridging.," *Br. Heart J.*, 1995; 74(1): 90–2.
68. K. Tsujita *et al.*, "Impact of Myocardial Bridge on Clinical Outcome After Coronary Stent Placement," *Am. J. Cardiol.*, 2009; 103(10): 1344–1348.
69. P. K. Haager, E. R. Schwarz, J. vom Dahl, H. G. Klues, T. Reffelmann, and P. Hanrath, "Long term angiographic and clinical follow up in patients with stent implantation for symptomatic myocardial bridging," *Heart*, 2000; 84(4): 403–408.
70. Broderick, Kereiakes, Whang, Toltzis, and Abbottsmith, "Myocardial Bridging May Predispose to Coronary Perforation During Rotational Atherectomy.," *J. Invasive Cardiol.*, Apr. 1996; 8(3): 161–163.
71. M. Zhang, W. C. Kang, C. Il Moon, S. H. Han, T. H. Ahn, and E. K. Shin, "Coronary Artery Perforation Following Implantation of a Drug-Eluting Stent Rescued by Deployment of a Covered Stent in Symptomatic Myocardial Bridging," *Korean Circ. J.*, 2010; 40(3): 148.
72. W. Li, Y. Li, L. Sheng, and Y. Gong, "Myocardial bridge: is the risk of perforation increased?," *Can. J. Cardiol.*, 2008; 24(11): e80–e81.
73. A. Tandar, B. K. Whisenant, and A. D. Michaels, "Stent fracture following stenting of a myocardial bridge: Report of two cases," *Catheter. Cardiovasc. Interv.*, 2008; 71(2): 191–196.
74. Derkacz A, Nowicki P, Protasiewicz M, Reczuch K, Szczepanik- Osadnik H, Witkowska M. [Multiple percutaneous coronary stent implantation due to myocardial bridgingda case report]. Kardiol Pol, 2007; 65: 684–7.
75. P. B. Kunamneni *et al.*, "Outcome of intracoronary stenting after failed maximal medical therapy in patients with symptomatic myocardial bridge," *Catheter. Cardiovasc. Interv.*, 2008; 71(2): 185–190.
76. M. T. Corban *et al.*, "Myocardial bridging: Contemporary understanding of pathophysiology with implications for diagnostic and therapeutic strategies," *J. Am. Coll. Cardiol.*, 2014; 63(22): 2346–2355.
77. V. J. de M. Lima, J. S. Cavalcanti, and T. Tashiro, "Myocardial Bridges and their Relationship to the Anterior Interventricular Branch of the Left Coronary Artery," *Arq. Bras. Cardiol.*, 2002; 79(3): 219–222.
78. X.-H. Huang *et al.*, "Surgical outcome and clinical follow-up in patients with symptomatic myocardial bridging.," *Chin. Med. J. (Engl).*, Sep. 2007; 120(18): 1563–6.
79. Q. Y. Wu and Z. H. Xu, "Surgical treatment of myocardial bridging: report of 31 cases," *Chin Med J*, 2007; 120(19): 1689–1693.
80. S. Attaran, M. Moscarelli, T. Athanasiou, and J. Anderson, "Is coronary artery bypass grafting an acceptable alternative to myotomy for the treatment of myocardial bridging?," *Interact. Cardiovasc. Thorac. Surg.*, 2013; 16(3): 347–349.
81. L. A. Bockeria, S. G. Sukhanov, E. N. Orekhova, M. P. Shatakhyan, D. A. Korotayev, and L. Sternik, "Results of coronary artery bypass grafting in myocardial bridging of left anterior descending artery," *J Card Surg*, 28(3): 218–221.
82. Z. Xu, Q. Wu, H. Li, and G. Pan, "Myotomy after previous coronary artery bypass grafting for treatment of myocardial bridging," *Circulation*, 2011; 123(10): 1136–1137.
83. R. C. Hill, W. R. Chitwood, T. M. Bashore, J. D. Sink, J. L. Cox, and A. S. Wechsler, "Coronary Flow and Regional Function before and after Supraarterial Myotomy for Myocardial Bridging," *Ann. Thorac. Surg.*, 1981; 31(2): 176–181.
84. N. D. Hillman, C. Mavroudis, C. L. Backer, and C. E. Duffy, "Supraarterial decompression myotomy for myocardial bridging in a child," *Ann. Thorac. Surg.*, 1999; 68(1): 244–246.
85. A. Crespo, J. I. Aramendi, G. Hamzeh, and R. Voces, "Off-pump supra-arterial myotomy for myocardial bridging," *Eur. J. Cardio-thoracic Surg.*, 2008; 34(3): 682–684.
86. A. M.B., V. E. F., and P. N., "Robotic-assisted surgical myotomy in a 27-year-old man with myocardial bridging of the left anterior descending coronary artery," *Interactive Cardiovascular and Thoracic Surgery*, 2010; 11(2): 185–187.
87. J. H. Boyd *et al.*, "Surgical Unroofing of Hemodynamically Significant Left Anterior Descending Myocardial Bridges," *Ann. Thorac. Surg.*, 2017; 103(5): 1443–1450.