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Case Report

Myocardial Bridging, A Neglected Anomaly In Acute Coronary Syndrome: Case Report

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ABSTRACT

Background: Myocardial Bridging (MB) is an anomaly presented by an intra-myocardial path of a part of one of the major coronary arteries. Functional MB is less commonly observed on angiography (0.5–16%) and range from 4 to 80 mm in length. This case report elaborates a symptomatic MB occurrence in a patient manifesting as an acute coronary syndrome.

Case presentation: A 66 years old female was presented to the emergency room (ER) with typical angina gradually increased since 1 week ago. Associated symptoms were dyspepsia, episodic syncope, and dyspnea. Revealed vital signs were within normal limits. The cardio-pulmonary examination was unremarkable. Electrocardiogram (ECG) showed inverted T waves on V1-V4, cardiac biomarkers were not increased, chest x-ray disclosed an enlarged heart, echocardiography showed left ventricular hypertrophy (LVH) with normal left ventricle ejection fraction (LVEF). Nitrate, aspirin, and P2Y12 inhibitor were then administered in the ER, and the patient was then transferred to catheterization lab. Coronary angiography displayed a MB in the middle left anterior descending (LAD), the patient was then treated with bisoprolol as a maintenance therapy.

Conclusion: MB, if presenting symptomatically, especially as an acute coronary syndrome (ACS), may become life threatening if not recognized and treated appropriately. Symptoms management and flow normalization in such circumstances are best achieved through drug therapy by using beta-blocker and revascularisation if the medicinal therapy were not improved.

1. Introduction

The epicardial surface of the heart was generally course along by the three major coronary arteries. On occasion, however, a small part of coronary artery descends into the myocardium for a variable distance. This abnormality, termed MB, occurs in 5% and up to 12% of patients and commonly occurs exclusively on the LAD artery. Although bridging is not thought to be of any hemodynamic significance in most cases, MB has been associated with ACS, coronary spasm, arrhythmia, syncope, depressed LV function, myocardial stunning, early death after cardiac transplantation, and sudden death. The prognosis of cases with MB, hence, is not as harmless as it was believed to be in the past.[1]

The following presented case is a fascinating specimen of the clinical findings of a symptomatic MB. Therefore, it is engaging not only because of the rarity of the case but also because it brings proper attention to an abnormality that is frequently neglected and overlooked.

2. Case Illustration

A 66 years old female was presented to ER with typical angina Canadian Coronary Score (CCS) class III which gradually increased since 1 week ago. Associated symptoms were dyspnea and dyspepsia. A week before admission, the patient was experiencing substernal chest pain and shortness of breath. From history taking, the patient had no cardiovascular risk at all and the symptoms were manifested for the first time.

On physical examination, blood pressure (BP) 110/70mmHg, pulse 86 BPM, respiration rate (RR) 26 breaths/minute, temperature 36.4C, oxygen saturation (SaO2) 97% room air. The cardio-pulmonary examination was unremarkable. ECG on admission showed T wave inversion on leads V1-V4 (Figure 1a). Briefly, minutes after admission the symptoms were progressively worsening and syncope occurred. Serial ECG showed T-wave evolution, prolonged

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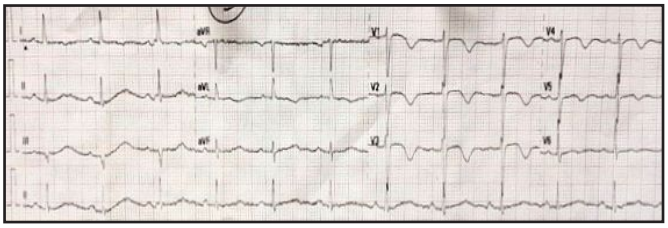

Figure 1a. ECG on admission showed: T- Inverted Lead V1-V4

QT-interval, and QTC 470ms, which was suggested associated with syncope (Figure 1b). Radiological examination of the thorax showed an enlarged heart. The echocardiography examination results were consistent of LVH with normal LVEF.

The patient was promptly monitored as a symptoms of ACS, free-flow oxygenation with nasal cannules were administered, and was given initial drug therapies with nitrate (sublingual isosorbide dinitrate 5mg, which could be repeated up to three times with five minutes interval between each administration), dual anti-platelet therapy (DAPT) which consisted of a loading dose of aspirin 80mg tablets up to a total of 320mg and P2Y12 inhibitor, in this case, clopidogrel 75mg was the one readily available up to a dose of 300mg.

The blood sample was then obtained for cardiac biomarkers exam, which showed no remarkable increased (troponin T-hs 3.0 pg/mL) and no disturbance of electrolytes (Table 1), the patient was then admitted to catheterization laboratory as clinical presentations and ECG findings lead to an impression of an occluded coronary artery and myocardial ischemia.

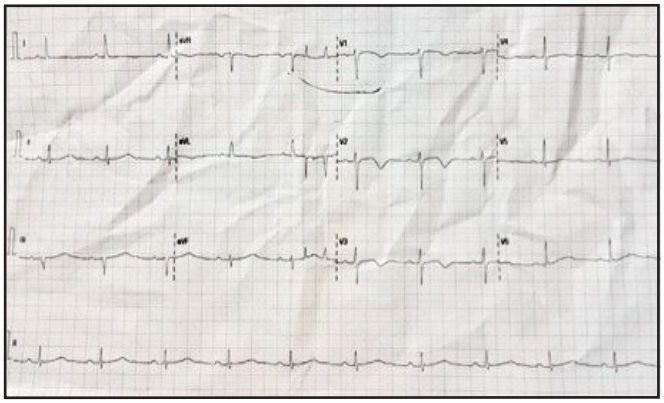

Figure 1b. ECG 2 (minutes after admission): T-wave evolution and long QT-interval (QTC 470ms)

Early coronary angiography showed presence of a systolic narrowing and recovered in diastolic phase in mid LAD (Figure 2a and 2b). There was no stenosis of the remaining coronary tracts that might cause ischemic symptoms. The patient was diagnosed with symptomatic MB. These findings also suggest that typical angina and ECG abnormalities that related to syncope occurance originate from this anomaly. Catheterization was done and the patient was treated with bisoprolol for maintenance therapy. After the patient was controlled with regular administration of bisoprolol, she did not experience symptoms anymore. The patient was discharged from the hospital with medications and no symptoms have been observed during follow-up at the outpatient clinic

Table 1. Laboratory Studies

Variables	Result	Reference Range
Hemoglobin	14.10 g/dL	11.70 – 15.50 g/dL
Hematocryte	40.90%	35-57%
Leucocyte	8.79 x 10 ^ 3 / uL	3.60 – 11.0 x 10 ^ 3 u/L
Thrombocyte	299 x 10 ^ 3 / uL	150.0 – 450.0 x 10 ^ 3 u/L
Creatinine	0.66 mg/dL	0.5 – 1.1 mg/dL
eGFR	92.1 mL/mnt/1.73 m ^ 2	
Random Blood Glucose	100 mg/dL	<200 mg/dL
Troponin T-hs	3.0 pg/mL	0 – 14.00 pg/mL
CK-MB	15.3 U/L	7-25 U/L
Sodium	140 mmol/L	136-145 mmol/L
Potassium	4 mmol/L	3.6 – 5.0 mmol/L
Chloride	101 mmol/L	90-107 mmol/L

3. Dsicussion

In this case, the patient was experiencing a symptomatic MB: ACS and syncope. Angina symptoms may occur because of the MB. When undergoing systolic phase, the epicardial artery within the myocardial bridge got forcibly closed due to myocardial contraction, disturbing the blood flow, thus resulting in an ischemic process and manifesting clinically as an acute coronary event.[2]

The clinical findings of MB should be understood to achieve an accurate diagnosis and the appropriate treatment. We review and elaborate our assessment in this case report, hoping that other centers that experience the same situation as ours, can recognize the problem promptly to be able to withstand this condition.

MB occurs when at least one of the coronary arteries passes through the heart muscle rather than lying on its surface.[3] MB exhibits a typical angiographic finding; the bridged segment (myocardium passes over the involved segment of coronary artery), known as tunneled, is on normal diameter during diastole and suddenly narrows during systole.[4] A narrowed epicardial artery during the systolic phase is the typical angiographic characteristic of a MB and often resolved in the diastolic phase of the cardiac cycle because it is not only an angiographic event but also 15% of coronary flow normally occurs during systole. So it needs a particular clinical condition for example, tachycardia, which can induce an ischemic symptoms on the MB caused by the diastolic phase being shortened and the importance of systolic blood flow increased.[5,6] “milking” is an effect of the external pressure of the myocardium on the artery. Myocardial ischemia triggering caused by reduced flow to the distal segment of the artery.[7] Hemodynamic abnormalities in MB characterized by a persistent decrease in diastolic vessel diameter, increase in blood flow velocities and retrograde flow, and a reduced flow reserve, leading MB is not only the diameter of the intramuscular segment smaller compared to the overall adjacent proximal segment, but also during diastole there is a persistent reduction of 34%–51% in the bridged segment.[8]

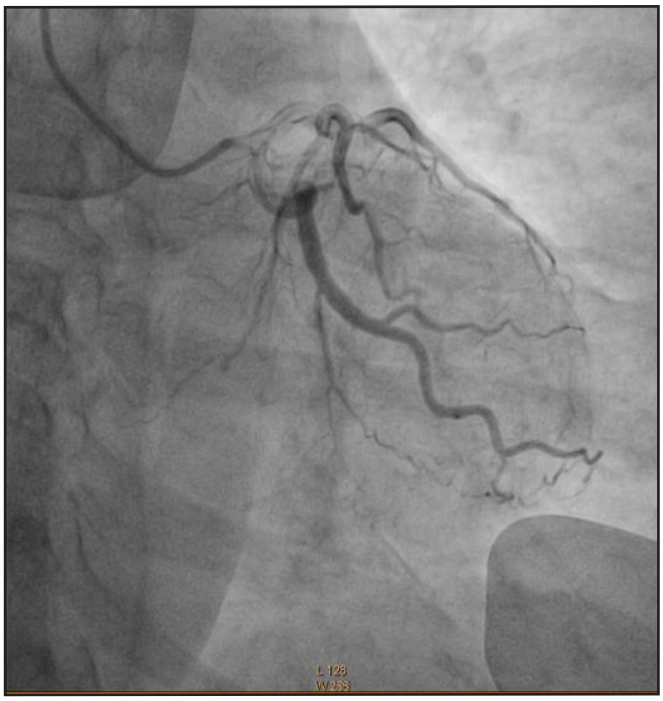

Figure 2a. There was no narrowing in the middle LAD segment in diastolic phase.

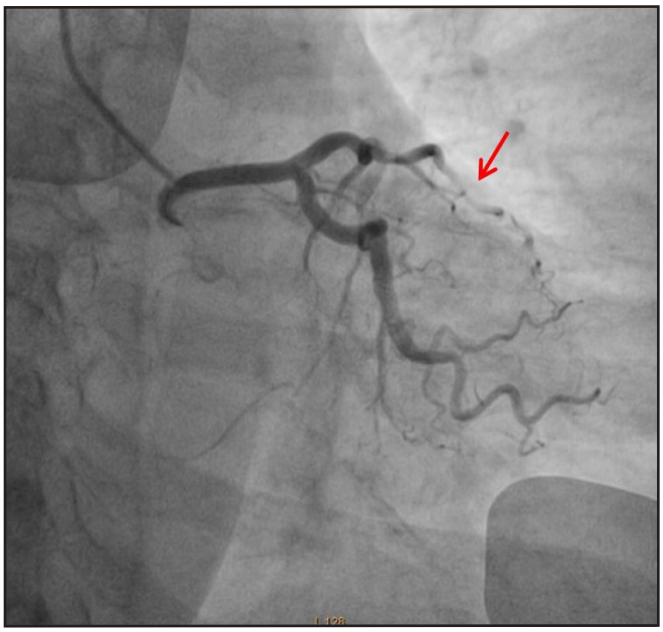

Figure 2b. Systolic phase showed MB on middle LAD.

Our patient was admitted to ER with typical crushing chest pain, consistent with ACS. 1 week before admission, the patient was already experiencing recurrent but mild chest discomfort and shortness of breath. From history taking, the patient had no cardiovascular risk at all and the symptoms manifested for the first time. In a view of the epidemiological prevalence of this anomaly, clinical suspicion of a MB would be warranted in all cases of typical or atypical chest pain in subjects who have a low probability of atherosclerosis because they are virtually free from cardiovascular risk factors.

Oxygenation was administered while 12-Łeads ECG is being prepared. The ECG result showed T wave inversion on leads V1-V4 that had an impression of myocardial ischemia. In the ER, the symptoms were progressively worsening and syncope occurred. A few minutes after syncope occured, serial ECG showed T-wave evolution, and a prolonged QT-interval. MB may induce several of ECG abnormalities, and one of them is prolonged QT-interval. The cardiac muscle of a patient with a long QT syndrome takes longer than usual to repolarize between beats. A prolonged QT-interval is a type of electrical disruption that can be noticed on an ECG. (AP mid-myocardial M cell > AP other myocardial cells) that resulted in a protracted TP-e Interval (distance between Peak and the end T-wave). Long QT-interval may be associated with syncope which occurred during admission and shortness of breath may occur because of a mismatch between ventilation and perfusion (V/Q mismatch).[13]

Another important clinical feature we should noticed that may be found in a case of MB is Wellens syndrome. A subset of patient with ischemic chest pain presents with deep arrowhead T wave inversions in multiple precordial leads (e.g., V1 or V2 to V4) with or without cardiac enzyme elevations and with minimal or no ST elevations. This pattern, called the Wellens syndrome or the LAD-T wave inversion pattern, is typically caused by high-grade stenosis of the LAD coronary artery system. These deep T inversions are the result of a delay in regional repolarization produced by ischemic injury.[14–16]

In our case, the narrowing of tunneled artery was proved by coronary angiography. The use of other imaging modalities to detect MB such as intravascular ultrasound (IVUS) and cardiac computed tomography (CCT) is possible even the milking effect or vessel changes are absent or mild. The coronary artery is shown on IVUS to allow precise measurements of lumen diameter and vessel wall thickness, which can be used for diagnostic criteria, by locating an echolucent area just between the bridging segment and the epicardial tissue, referred to as the "half-moon phenomenon". The other assessment of MB is using CCT, a noninvasive technique performed on an outpatient basis that can visualize the lumen of the coronary, the vessel, and the myocardial wall with a 3-dimensional image.[6,9] Diastolic flow anomalies have been found in patients with MB using intracoronary Doppler investigations.

Coronary angiography revealed no atherosclerotic lesion that might cause symptomps of ACS in our patient. However, based on few report of the other contributes to symptomatic MB beside narrowing epicardial artery during the systolic phase is the symptomps of MB can caused by processed of plaque formation at the entrance of a segment proximal to MB.[8] That segments susceptible become main factor in the distribution of atherosclerosis due to disturbed near-wall blood flow patterns. This pattern of blood flow is referred to wall shear stress (WSS). Coronary artery portions promptly proximal to the MB where the WWS is relatively lower, have structurally dysfunctional, flat and polygon-shaped endothelial cells. Whereas, endothelial cells in the bridged segments, where the WWS is on physiological levels or even higher, are structurally unbroken. Specifically, constriction within the bridged segment and severe vessel angulation at the junction of the bridged segment result in a heterogeneously diverse stress field within the proximal portion from the bridged segment. On the other hand, the intima of the tunneled segment is much thinner than the proximal segment, and it contains a higher proportion of the "contractile" subtype of smooth muscle cells, which is hypothesized to be related with the development of atherosclerotic lesions.[10]

Other pathophysiological changes can lead to the manifestations of myocardial ischemia in previously asymptomatic patients. For one, the worsening of the LV diastolic dysfunction associated with degenerative process, high blood pressure, and coronary atherosclerosis can aggravate the supply-demand mismatch imposed by myocardial bridging. The results of echocardiography examination in our case wereconsistent of LVH with normal LVEF. Another proposed hypothesis

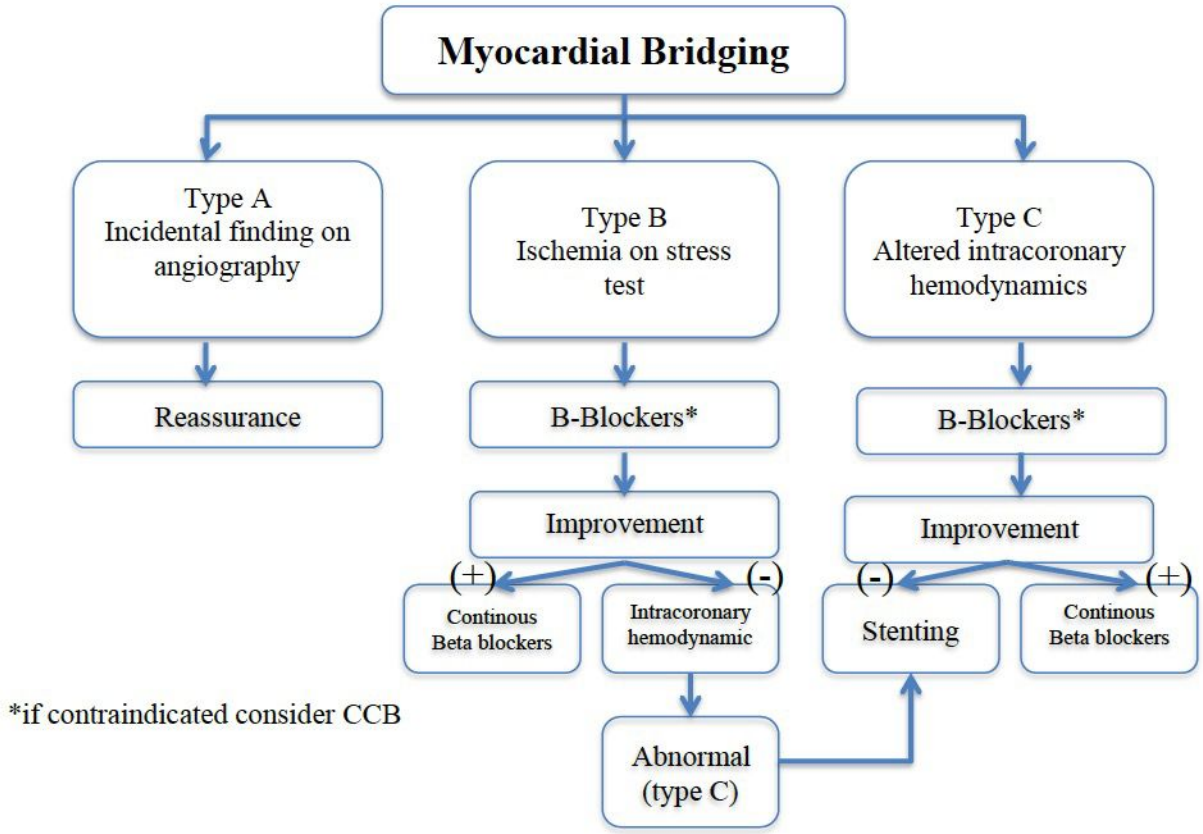

Figure 3. The Schwarz classification

elaborates that the development of LV hypertrophy can worsen the constriction inside the bridged segments and subsequently diminish the coronary microvascular reserve. Within the bridged segments, elevated mechanical pressure expectedly contributes to a constrictive vascular remodeling process as an effort to reinstate the intravascular load back to homeostatic levels. Lastly, the negative remodeling within the bridge can dwindle myocardial blood flow. Each of these factors could possibly contribute to a varying degree of severity to the development of symptoms in patients with MB.[10,11]

Symptomatic MB patients must be treated, medical therapy should include optimal doses of beta-blockers, CCB, and antiplatelet agents to relieve symptoms and signs myocardial ischemia and/or protecting against the risk of future coronary events.[17] In our case, patient was given bisoprolol for maintenance therapy. Beta-blockers remain as the treatment of choice to treat the hemodynamic disturbances brought about by the myocardial bridging, by decreasing the heart rate, increasing the diastolic coronary filling duration, diminishing contractility and constriction of the coronary arteries, therefore beta-blockers agents may cause improvement as found in our patient. If the patient has a cardiovascular risk factor, aggressive risk factor modification is strongly encouraged and antithrombotic drugs should be considered to be administered in patients with MB due to their increased risk of developing atherosclerosis.[5] One assessment modality to determine the need for antiplatelet therapy would be to perform a multislice computed tomography (MSCT) to identify subclinical coronary atherosclerosis.[18] CCB has been less often used in these patients. They may be particularly beneficial when beta-blocker therapy is contraindicated or when coronary vasospasm is suspected as a primary treatment. Nitrates may be used, but they have the potential to worsen symptoms due to their ability to reduce preload by intensifying systolic compression of the bridged segment, relieve vasospasm proximal to the bridge, and have an effect on systemic vasodilation, resulting in excessive contraction due to secondary tachycardia, exacerbating retrograde flow in the proximal segment, lowering coronary pressure distal to the bridged segment.[8,17] Vasodilators should therefore be avoided unless considerable coronary vasospasm is present.

Revascularization of the MB via stenting and surgery of the intracoronary artery have been tried in a few patients who experience symptomatic recurrence after medical therapy, but the results have been mixed.[19] The common surgical approaches are coronary artery bypass grafting (CABG) and myotomy. CABG is strongly recommended and beneficial in an extensive (>25 mm length) or deep (>5 mm depth) myocardial bridging. In the absence of lengthy or deep bridges, surgical myotomy should be considered for those at high risk of myocardial infarction, ventricular tachycardia, or resuscitated cardiac arrest, as well as patients with refractory symptoms despite medicinal therapy.[6]

The Schwarz classification (Figure 3) can serve as a guiding tool for directing therapeutic measures aimed for patients with MB due to it's correlation to relatively acceptable clinical outcomes after pharmacological and invasive interventions. Patients categorized within the Schwarz type A group does not need any treatment, whereas patients included within the type B and C groups show significant symptomatic improvement with beta-blockers or calcium channel blockers (CCB) after a five-year follow-up. Moreover, patients who fell within the aforementioned Schwarz type C group (who experience symptomatic recurrence after medical therapy) may be considered to undergo revascularization of the MB.[5]

4. Conclusion

MB is often asymptomatic. if presenting symptomatically, especially as ACS or syncope, MB may become lethal. Not all typical angina symptom is produced by atherosclerotic plaque of the epicardial coronary vessel. Symptomatic MB should be optimally treated by beta blocker agent to abolish symptoms and considering revascularization (Stenting/ Coronary artery bypass grafting (CABG) to normalize coronary flow if the medicinal therapy were not improved.

5. Declarations

5.1. Ethics Approval and Consent to participate
Not applicable.

5.2. Consent for publication
Not applicable.

5.3. Availability of data and materials
Data used in our study were presented in the main text.

5.4. Competing interests
Not applicable.

5.5. Funding source
Not applicable.

5.6. Authors contributions
Idea/concept: GPH, ASSM. Design: GPH. Control/supervision: ASSM. Data collection/processing: GPH. Extraction/Analysis/interpretation: ASSM, GPH, GRTH, DDS, ANK. Literature review: GPH, GRTH, DDS, ANK. Writing the article: GPH, GRTH, DDS, ANK. Critical review: ASSM, GPH, GRTH, DDS, ANK. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

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References

1. Ripa C, Cristina Melatini M, Olivieri F, Antonicelli R. Myocardial bridging: A “forgotten” cause of acute coronary syndrome-a case report. Int J Angiol. 2007;16(3):115–8.

2. Yuan SM. Myocardial bridging. Brazilian J Cardiovasc Surg. 2016;31(1):60–2.

3. He X, Ahmed Z, Liu X, Xu C, Zeng H. Recurrent attack of acute myocardial infarction complicated with ventricular fibrillation due to coronary vasospasm within a myocardial bridge: A case report. BMC Cardiovasc Disord. 2020;20(1):1–5.

4. Möhlenkamp, Stefan & Hort, Waldemar & Ge, Junbo & Erbel R. Update on myocardial bridging. Circulation. 2002;106(2616):22.

5. Corban MT, Hung OY, Eshtehardi P, Rasoul-Arzrumly E, McDaniel M, Mekonnen G, et al. Myocardial bridging: Contemporary understanding of pathophysiology with implications for diagnostic and therapeutic strategies. J Am Coll Cardiol. 2014;63(22):2346–55.

6. Tarantini G, Migliore F, Cademartiri F, Fraccaro C, Iliceto S. Left Anterior Descending Artery Myocardial Bridging: A Clinical Approach. J Am Coll Cardiol. 2016;68(25):2887–99.

7. Lozano Í, Baz JA, López Palop R, Pinar E, Picó F, Valdés M, et al. Long-term prognosis of patients with myocardial bridge and angiographic milking of the left anterior descending coronary artery. Rev Esp Cardiol. 2002;55(4):359–64.

8. Lee MS, Chen CH. Myocardial Bridging: An Up-to-Date Review. J Invasive Cardiol. 2015;27(11):521–8.

9. Nakaura T, Nagayoshi Y, Awai K, Utsunomiya D, Kawano H, Ogawa H, et al. Myocardial bridging is associated with coronary atherosclerosis in the segment proximal to the site of bridging. J Cardiol [Internet]. 2014;63(2):134–9. Available from: http://dx.doi.org/10.1016/j.jjcc.2013.07.005

10. Chatzizisis YS, Giannoglou GD. Myocardial bridges are free from atherosclerosis: Overview of the underlying mechanisms. Can J Cardiol. 2009;25(4):219–22.

11. Corban MT, Hung OY, Eshtehardi P, Rasoul-Arzrumly E, McDaniel M, Mekonnen G, et al. Myocardial Bridging. J Am Coll Cardiol [Internet]. 2014 Jun;63(22):2346–55. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0022202X15370834

12. Krasniqi X, Çitaku H. Anomalous Origin of Coronary Arteries. In: Vascular Biology - Selection of Mechanisms and Clinical Applications [Internet]. IntechOpen; 2020. Available from: https://www.intechopen.com/books/vascular-biology-selection-of-mechanisms-and-clinical-applications/anomalous-origin-of-coronary-arteries

13. Sarkar M, Niranjan N, Banyal P. Mechanisms of hypoxemia. Lung India [Internet]. 2017;34(1):47. Available from: http://www.lungindia.com/text.asp?2017/34/1/47/197116

14. Ambesh P, Sharma D, Kapoor A, Hess A-T, Shetty V, Hollander G, et al. Unusual Sign from an Unusual Cause: Wellens’ Syndrome due to Myocardial Bridging. Case Reports Cardiol. 2018;2018:1–3.

15. Pederçole GL, Oliveira MDP, Moritz MA, Cury RBB, Caixeta A. Myocardial bridging mimicking Wellens’ syndrome. J Xiangya Med. 2020;5:6–6.

16. Goldberger AL, Goldberger ZD, Shvilkin A. Myocardial Ischemia and Infarction, Part II. Goldberger’s Clin Electrocardiogr. 2018;92–103.

17. Bourassa MG, Butnaru A, Lespérance J, Tardif JC. Symptomatic myocardial bridges: Overview of ischemic mechanisms and current diagnostic and treatment strategies. J Am Coll Cardiol. 2003;41(3):351–9.

18. Toth PP. Subclinical atherosclerosis: What it is, what it means and what we can do about it. Int J Clin Pract. 2008;62(8):1246–54.

19. Ge J. New signs characteristic of myocardial bridging demonstrated by intracoronary ultrasound and Doppler. Eur Heart J [Internet]. 2000 Dec;20(23):1707–16. Available from: https://academic.oup.com/eurheartj/article-lookup/doi/10.1053/euhj.1999.1661