OPEN ACCESS
Scholars Bulletin
Abbreviated Key Title: Sch Bull
ISSN 2412-9771 (Print) |ISSN 2412-897X (Online)
Scholars Middle East Publishers, Dubai, United Arab Emirates
Journal homepage: https://saudijournals.com/sb

Subject Category: Internal Medicine

Myocardial Perfusion Imaging (MPI) as an Assessment Tool for Coronary Haemodynamics in A Patient with Myocardial Bridging of the Right Coronary Artery at Universitas Academic Hospital, South Africa; A Case Discussion

Alikor CA, Otto A

Department of Internal Medicine, University of Port Harcourt Teaching Hospital, Port Harcourt, Nigeria

DOI: 10.36348/sb.2020.v06i03.002 | **Received:** 24.02.2020 | **Accepted:** 04.03.2020 | **Published:** 11.03.2020

***Corresponding author:** Alikor CA

Abstract

We report a case of assessment of haemodynamics of myocardial bridging (MB) of the right coronary artery using myocardial perfusion imaging (MPI). Although most patients with MB are asymptomatic, some may present with ischemia and related complications, including myocardial stunning, infarction, arrhythmia, AV block, transient LV dysfunction and sudden death and hence it becomes necessary to evaluate the hemodynamics of these patients with MB whether symptomatic or not using MPI which is effective and noninvasive. The index case was a 61 year old man found to have myocardial bridging of the right coronary artery. He had no preceding symptom of ischaemia. Myocardial perfusion imaging however established the presence of inferior wall ischaemia in the absence of any coronary artery lumen narrowing.

Keywords: Myocardial Perfusion Imaging, Assessment Tool, Coronary Haemodynamics, Myocardial Bridging, Right Coronary Artery.

INTRODUCTION

Myocardial bridging is a congenital anomaly, an anatomical variation which occurs if a segment of a coronary artery travels through the myocardium instead of the myocardium surface. In MB, a coronary artery takes a course within a segment of the myocardium that compresses the lumen during ventricular systole despite a normal appearance during diastole. It has also been described as tunneled artery. During systole, this segment of the vessel is compressed, a condition referred to as milking or systolic "myocardial bridging"[1-2].

This phenomenon was first recognized more than 200 years ago [3], was first reported in depth in 1951, and was recognized angiographically in 1960. Myocardial bridging was first defined at autopsy by Reyman [4] in 1737. Later, Portmann and Iwig angiographically described temporary occlusion in a segment of the left anterior descending artery (LAD) during the systolic phase. In 1976, Noble *et al.*[5] detected this temporary occlusion in selective coronary angiography in 27 (0.5%) of 5,250 patients and named it the "milking effect." In their studies of autopsies, Ferreira *et al.* [6] distinguished between two types of bridging: superficial bridges (75% of cases) and deep bridges (25% of cases). However, there are no certain depth criteria set to classify myocardial bridges, and they are classified according to the routes that muscle bundles forming the myocardial bridges follow. Other than these routes, arterial segments may also be located in a deep interventricular gorge. The surface is not fully covered by myocardial fibers but rather by a thin layer of connective tissue, nerves, and fatty tissue. This kind of bridging, which is defined as incomplete, may also show compression in the systolic phase [7].

The degree of bridge segment narrowing during systole can be quantified as the percentage of the normal diameter of the artery immediately proximal to the MB [systolic diameter of the artery directly distal to bridge segment minus systolic diameter of the bridged segment)/systolic diameter of the artery directly distal

to bridging 9 100%]. The systolic narrowing was graded as severe ([75%), moderate (50 to 75%) and mild (>50%) systolic narrowing according to Noble grading [8].

On angiography, bridging is recognized as compression of a segment of a coronary artery during systole, resulting in narrowing that reverses during diastole. The dynamic and phasic nature of the obstruction serves to differentiate bridging from fixed coronary stenosis. Muscle bridges are more common in men than in women and tend to affect patients in their fourth decade of life [9].

The reported prevalence of bridging varies according to the method of evaluation. Pathologic studies have found a mean frequency of myocardial bridging of 25 percent (range 5 to 86 percent). Angiographic studies have reported prevalence of myocardial bridging to be 1.7 percent (range 0.5 to 16 percent). MB have also been found to be mainly confined to the Left Anterior Decending artery (LAD) (anterior interventricular branch of the left coronary artery) (picture 1) [1-2, 10-11] and rarely involves the right marginal branch of the left coronary artery (Rmd), the circumflex branch of the left coronary artery (RCX) and the right coronary artery. Patients with hypertrophic cardiomyopathy and recipients of cardiac transplants have shown high prevalence of MB.

The site, length, and severity of bridging, and resultant coronary stenosis, vary from patient to patient and may vary in the same patient from one examination to another. As an example, nitroglycerin augments the severity of compression, probably by reflex sympathetic stimulation of contractility and/or a lower intraluminal pressure in the coronary artery [12].

The degree of bridging is usually neither extensive nor severe. However, there is again a large degree of variability among patients:

- Bridging may involve a large segment of the vessel, extending from the origin to more than two-thirds of the vessel length, thereby jeopardizing the flow to secondary branches.
- In some cases, bridging is so severe that there is almost complete obliteration of the vessel lumen during systole.

Bridging can involve the secondary branches, especially the septal perforators. Systolic compression of septal perforators appears to be more common in patients with hypertrophic cardiomyopathy, in whom it has been postulated to be one of several mechanisms responsible for myocardial ischemia

It affects men more than women (2:1 ratio) and manifests clinically around 40 to 50 years of age [13, 14]. Although MB was generally thought to be a benign variation, however, cumulative evidence now suggests that it may be associated with myocardial ischemia[15] myocardial infarction [16], arrhythmias, and sudden death[17] Therefore, it has become important for clinical doctors to evaluate the hemodynamics of MB.

The crucial point is the coronary circulation physiopathology during the cardiac cycle. The arterial segment running within the myocardial wall is actually squeezed, undergoing calibre narrowing, during systole2. Even though coronary flow is mainly diastolic, arterial compression with over 70% arterial lumen reduction, causing myocardial ischemia, even leading to Acute Myocardial Infarction (AMI), was found out on selective coronary angiography [13, 18]. Some Authors [19, 21] have considered this anomaly as the cause of tachyarrhythmias. Myocardial bridge was the only abnormality found out in 4% of sudden cardiac death reports of an otherwise normal coronary tree [19-21].

Several methods, such as intravascular ultrasound (IVUS), intracoronary Doppler, and intracoronary pressure, have been utilized to measure both anatomical and physiological parameters of coronary vessels. Unfortunately, all these techniques are either invasive and/or not routinely performed.

The noninvasive method, myocardial perfusion imaging (MPI) is widely accepted to assess myocardial ischemia and prognosis in patients with known or suspected coronary artery disease, yet few patients with MB have been evaluated with such nuclear medicine techniques [22-25].

Cardiac literature does not propose established standards for dealing with myocardial bridging. Pharmacological treatment is applied most frequently [23-24] but also percutaneous transluminal coronary angioplasty [23-25] and cardiosurgical interventions (myotomy, aortal-coronary bypassing) [27, 28] have been practiced.

Myocardial bridging and cardiac ischemia

Effects on coronary blood flow — Myocardial bridging, which causes coronary artery obstruction only during systole, would not be expected to significantly reduce total myocardial perfusion significantly since almost two-thirds of blood flow in the left coronary system occurs in diastole, most of which is directed to the subendocardial layer. There are, however, two important considerations:

- Frame by frame analysis of cine coronary angiograms has shown a "spill over" phenomenon that is characterized by persistence of narrowing in early diastole. In general, the more severe the

systolic narrowing is, the more likely that spill over will occur.
- With tachycardia, there is more systolic compression and systole occupies a greater percentage of the cardiac cycle because of shortening of diastolic filling period.

These mechanisms may explain why several studies of rapid atrial pacing or pharmacologic stimulation with dobutamine detected evidence of myocardial ischemia in patients with isolated systolic bridging of the LAD. It should be cautioned, however, that pacing stress may differ from exercise stress: there is a progressive decrease in systolic blood pressure with pacing versus a progressive increase with exercise. The elevation in intracoronary systolic pressure with exercise may help to prevent compression. There are no comparative data between pacing and exercise to test this hypothesis. The use of adenosine, which does not increase contractility or cause significant tachycardia, may underestimate ischemia potentially related to myocardial bridging.

Quantitative coronary angiography has been combined with intravascular ultrasound (IVUS) and Doppler flow measurements in patients with isolated bridging of the proximal LAD [1, 10-11]. The following observations were noted in two reports from the same center [10, 11].
- A specific, echolucent half-moon phenomenon over the bridge segment, which exists throughout the cardiac cycle.
- A characteristic systolic compression (either concentric or eccentric) with delayed relaxation in diastole of the myocardial bridging segment.
- Accelerated flow at early diastole (finger-tip phenomenon) with either no or reduced systolic antegrade flow (figure 1).
- Decreased diastolic/systolic velocity ratio.
- Retrograde flow in the proximal segment, which is provoked and enhanced by nitroglycerin injection.
- The cross-sectional lumen area variation in one report was 40±25 percent in the bridging segments versus 9±7 percent in the normal segments. Atherosclerotic lesions occurred proximal to, but not within or distal to the bridging segments.
- A highly characteristic pattern showing a prominent peak in coronary velocity in early diastole was seen in 86 percent of patients (figure 1).
- The coronary flow velocity reserve ratio (peak/resting ratio) was 2.2, which is below normal, possibly due to delayed release of bridging and/or proximal atherosclerosis; this finding may explain signs of ischemia in some of these patients.

Fractional flow reserve (FFR) was measured at baseline and during dobutamine infusion in 12 patients with myocardial bridging [29]. The mean and diastolic FFR decreased from baseline with dobutamine, with the diastolic change being more prominent. The authors postulated that overshooting of systolic pressure interferes with and is a cause of error in FFR measurement based upon mean pressures, while the diastolic FFR appears to be the method of choice. Other reports have emphasized the use of dobutamine rather than adenosine when assessing myocardial bridging with FFR [30].

CASE REPORT

61 year old man, not previously diagnosed hypertensive or diabetic who on regular exercise at the gym discovered that his radial pulse was irregular. There was no associated angina, orthopnoea, or paroxysmal nocturnal dyspnoea. No peripheral oedema. No history of transient ischaemic attack or strokes. No history of previous deep veinous thrombosis bilaterally. Uses bicycle daily without any symptoms.

Risk factor: Father died at 50 years of age and there is a positive family history of ischaemic heart disease. No surgical history. The general physical examinations were essentially normal. Cardiovascular system examination revealed the following: Pulse 70 b/m, regular and peripheral pulses were normal Blood pressure was 130/100mmHg and heart sounds were normal. Other systems were normal.

Regular ECG- Showed sinus rhythm. Stress ECG- at the peak of exercise, ST segment depression was noted in the infero-lateral leads, but no chest pain was reported.

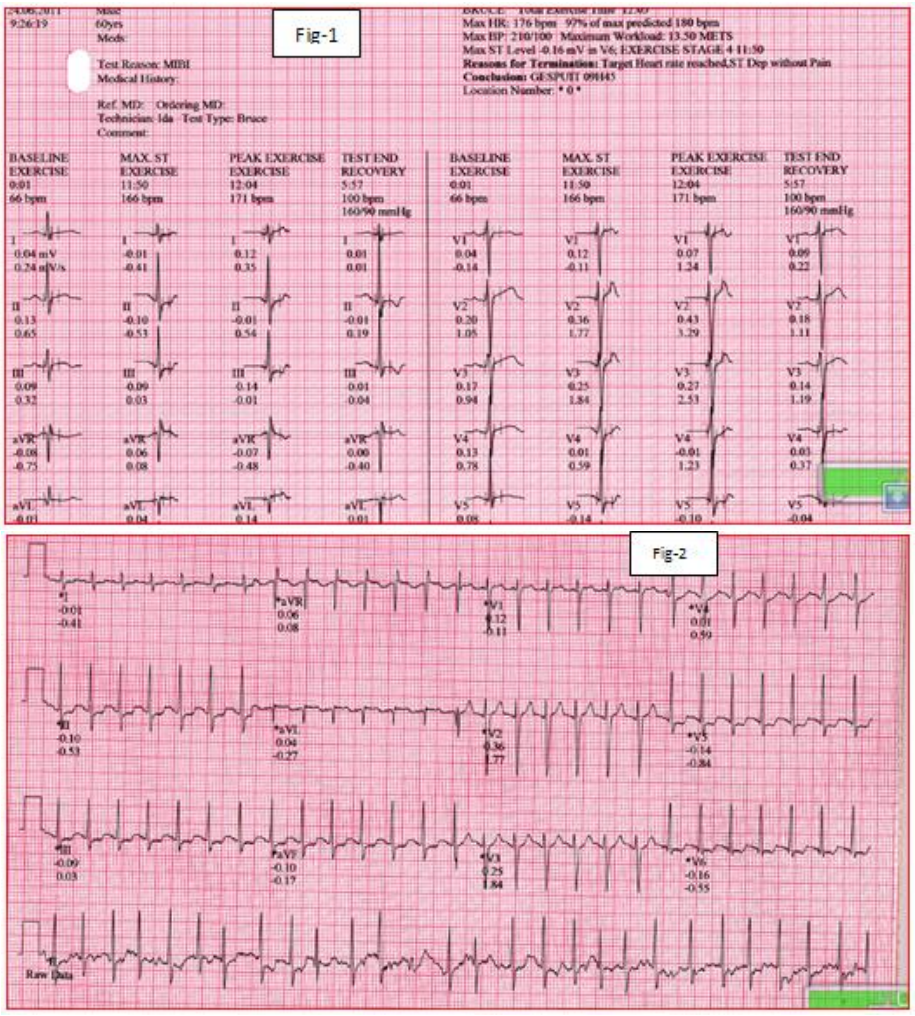

Fig-1 & 2: Stress ECG of index case

Exercise Stress MIBI Myocardial Perfusion Imaging- A maximum heart rate of 176 bpm (97% of age predicted maximum) was reached after 12 minutes (13.5 METS). The perfusion images showed an inferior wall defects. The gated study showed normal left ventricular wall motion. The LVEF was 66% and the EDV was 109ml. The conclusion was that there was evidence of inferior wall myocardial ischaemia demonstrated on perfusion imaging and the left ventricular function was normal.

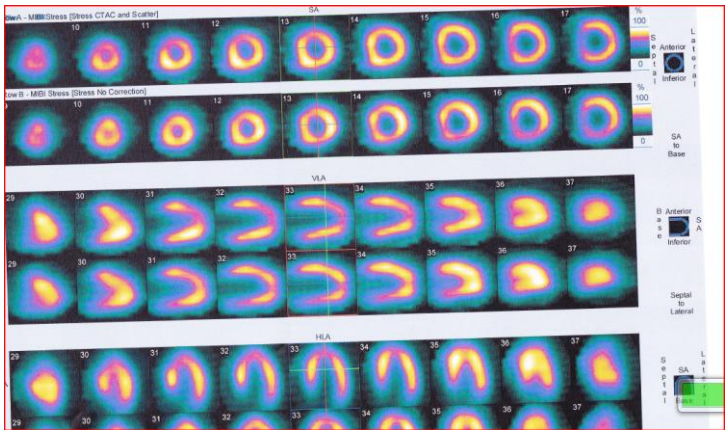

Fig-2: Perfusion image of index case

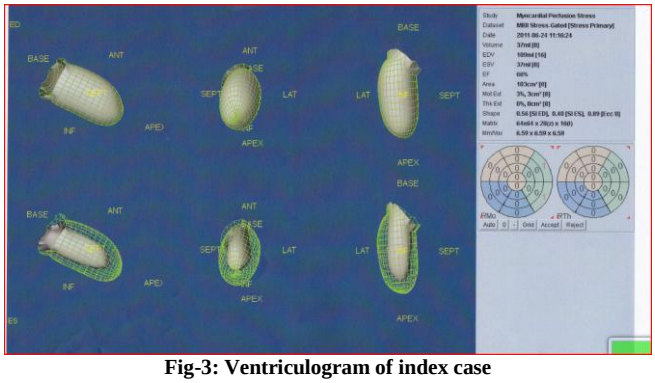

Fig-3: Ventriculogram of index case

Echocardiography (heart sonar) revealed that the ventricular walls were normal with a LVEF of 59% and some mild left ventricular hypertrophy. CT Coronary angiogram showed bridging of the right main coronary artery with decreased flow during systole. No coronary artery lumen narrowing was seen.

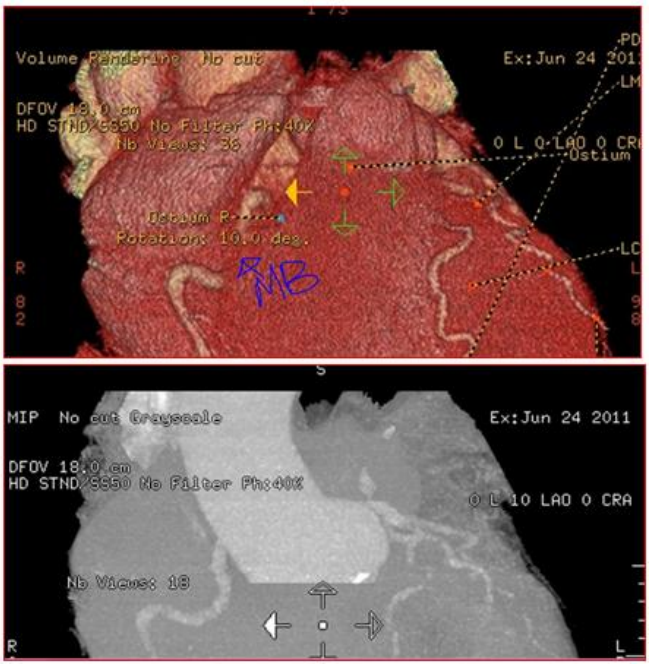

Fig-4: CT Coronary angiogram of index case

DISCUSSION

A myocardial SPECT perfusion imaging is a noninvasive and well-validated method presenting a radiopharmaceutical uptake by cardiomyocytes. The uptake is in wide range proportional to myocardial blood flow.

MB is usually considered a relatively benign congenital coronary anomaly that most commonly affects the mid-portion of the LAD [1]. In our patient it was found in the main right coronary artery which is a rare presentation. However, there is increasing evidence of its association with various clinical manifestations such as myocardial ischemia, infarctions, and sudden cardiac death. Thus, as a noninvasive functional examination, MPI could provide pertinent hemodynamic information needed to evaluate the clinical significance of MB.

The majority of patients with isolated MB do not have negative clinical relevance. However, in patients with MB, especially those who have a maximum systolic reduction of more than 75%, MPI should be performed to check for incidents of myocardial ischemia. The myocardial ischemia induced by MB is associated more closely with the degree of systolic narrowing.

Our patient presented asymptomatically. However stress ECG showed some evidence of ischaemia at the peak of exercise which was further demonstrated by the MPI. The possible reason for the Stress-ECG associated evidence of ischaemia may be due to the decreased flow during systole as reported by the coronary angiogram. However, the decreased systolic flow or narrowing of the systolic blood flow may have been in the mild catergory in view of the absence of clinical symptoms of ischaemia. This agrees

with the findings of Kun Tang *et al*. in china, were they studied the role of myocardial perfusion imaging in evaluating patients with myocardial bridging and they concluded on the note that myocardial ischaemia that resulted from bridging is associated more closely with the degree of systolic narrowing[31].

Rafał Gawor *et al*. [32] in their study of gated SPECT MPI among 42 patients with MB demonstrated that the incidence and severity of myocardial perfusion abnormalities in patients with myocardial bridges correlates with the degree of systolic constriction of the coronary artery. In the studied group of patients, regional perfusion abnormalities were observed in over 40% of individuals with bridges narrowing arteries by 50% or more.

Those perfusion abnormalities were stress induced, usually of mild or moderate intensity. This further buttresses the importance of assessment for ischaemia in patients with MB using the only available noninvasive technique MPI.

CONCLUSION

The application of MPI in evaluating MB has important clinical implications. It can provide us not only with the hemodynamic information of the bridging segment but also with valuable information as to how to proceed with the treatments for patients with MB. (1) there is no need for therapy in patients with systolic narrowness less than 75%; (2) in patients with severe symptoms and systolic narrowness more than 75%, MPI should be performed to determine whether there is myocardial ischemia present before further treatment is planned.

REFERENCE

1. Möhlenkamp, S., Hort, W., Ge, J., & Erbel, R. (2002). Update on myocardial bridging. *Circulation*, *106*(20), 2616-2622.
2. Alegria, J. R., Herrmann, J., Holmes Jr, D. R., Lerman, A., & Rihal, C. S. (2005). Myocardial bridging. *European heart journal*, *26*(12), 1159-1168.
3. Reyman, H.C. (1737). Disertatis de vasis cordis propiis. *Bibl Anat*, 2:366.
4. Reyman, H.C. (1737). Diss. de vasis cordis propriis. *Bibl Anat*, 2:359–379
5. Noble, J., Bourassa, M.G., Petitclerc, R., Dyrda, Y.(1976). Myocardial bridging and milking effect of the left anterior descending coronary artery: normal variant or obstruction? *Am J Cardiol*, 37:993–999
6. Ferreira, A.G, Jr., Trotter, S.E., Konig, B.Jr., Decourt, L.V., Fox, K., Olsen, E.G.(1991). Myocardial bridges: morphological and functional aspects. *Br Heart J*, 66:364–367
7. Möhlenkamp, S., Hort, W., Ge, J., & Erbel, R. (2002). Update on myocardial bridging. *Circulation*, *106*(20), 2616-2622.
8. Noble, J., Bourassa, M. G., Petitclerc, R., & Dyrda, I. (1976). Myocardial bridging and milking effect of the left anterior descending coronary artery: normal variant or obstruction?. *American Journal of Cardiology*, *37*(7), 993-999.
9. Angelini, P. Trivellato M, Donis J, Leachman RD: Myocardial bridges: A review. *Progr Cardiovasc Dis 1983; 26: 75*, *88*.
10. Alegria, J. R., Herrmann, J., Holmes Jr, D. R., Lerman, A., & Rihal, C. S. (2005). Myocardial bridging. *European heart journal*, *26*(12), 1159-1168.
11. Arnau, M. V., Martínez, L. D., Almenar, L. B., Lalaguna, L. A., Ten, F. M., & Palencia, M. P. (1999). Myocardial bridging as a cause of acute ischemia. Description of a case and review of the literature. *Revista espanola de cardiologia*, *52*(6), 441-441.
12. Yano, K., Yoshino, H., Taniuchi, M., Kachi, E., Shimizu, H., Watanuki, A., & Ishikawa, K. (2001). Myocardial bridging of the left anterior descending coronary artery in acute inferior wall myocardial infarction. *Clinical cardiology*, *24*(3), 202-208.
13. Hongo, Y., Tada, H., Ito, K., Yasumura, Y., Miyatake, K., & Yamagishi, M. (1999). Augmentation of vessel squeezing at coronary-myocardial bridge by nitroglycerin: study by quantitative coronary angiography and intravascular ultrasound. *American heart journal*, *138*(2), 345-350.
14. Angelini, P. Trivellato M, Donis J, Leachman RD: Myocardial bridges: A review. *Progr Cardiovasc Dis 1983; 26: 75*, *88*.
15. Berry, J. F., von Mering, G. O., Schmalfuss, C., Hill, J. A., & Kerensky, R. A. (2002). 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*, *56*(1), 58-63.
16. Ge, J., Jeremias, A., Rupp, A., Abels, M., Baumgart, D., Liu, F., ... & Erbel, R. (1999). New signs characteristic of myocardial bridging demonstrated by intracoronary ultrasound and Doppler. *European Heart Journal*, *20*(23), 1707-1716.
17. Ge, J., Erbel, R., Rupprecht, H. J., Koch, L., Kearney, P., Görge, G., ... & Meyer, J. (1994). Comparison of intravascular ultrasound and angiography in the assessment of myocardial bridging. *Circulation*, *89*(4), 1725-1732.
18. Haager, P. K., Schwarz, E. R., Vom Dahl, J., Klues, H. G., Reffelmann, T., & Hanrath, P. (2000). Long term angiographic and clinical follow up in patients with stent implantation for

symptomatic myocardial bridging. *Heart*, *84*(4), 403-408.
19. Ferreira, A. G., Trotter, S. E., König, B., Decourt, L. V., Fox, K., & Olsen, E. G. (1991). Myocardial bridges: morphological and functional aspects. *Heart*, *66*(5), 364-367.
20. Erbel, R., Rupprecht, H. J., GE, J., Gerber, T., Görge, G., & Meyer, J. (1993). Coronary Artery Shape and Flow Changes Induced by Myocardial Bridging: Assessment by Intravascular Ultrasound. *Echocardiography*, *10*(1), 71-77.
21. Ge, J., Erbel, R., Rupprecht, H. J., Koch, L., Kearney, P., Görge, G., ... & Meyer, J. (1994). Comparison of intravascular ultrasound and angiography in the assessment of myocardial bridging. *Circulation*, *89*(4), 1725-1732.
22. Escaned, J., Cortés, J., Flores, A., Goicolea, J., Alfonso, F., Hernández, R., ... & Macaya, C. (2003). Importance of diastolic fractional flow reserve and dobutamine challenge in physiologic assessment of myocardial bridging. *Journal of the American College of Cardiology*, *42*(2), 226-233.
23. Hakeem, A., Cilingiroglu, M., & Leesar, M. A. (2010). Hemodynamic and intravascular ultrasound assessment of myocardial bridging: fractional flow reserve paradox with dobutamine versus adenosine. *Catheterization and Cardiovascular Interventions*, *75*(2), 229-236.
24. Mouratidis, B., Lomas, F. E., & McGill, D. (1995). Thallium-201 myocardial SPECT in myocardial bridging. *Journal of Nuclear Medicine*, *36*(6), 1031-1033.
25. Vallejo, E., Morales, M., Sánchez, I., Sánchez, G., Alburez, J. C., & Bialostozky, D. (2005). Myocardial perfusion SPECT imaging in patients with myocardial bridging. *Journal of nuclear cardiology*, *12*(3), 318-323.
26. American Heart Association Writing Group on Myocardial Segmentation and Registration for Cardiac Imaging:, Cerqueira, M. D., Weissman, N. J., Dilsizian, V., Jacobs, A. K., Kaul, S., ... & Verani, M. S. (2002). Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart: a statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. *Circulation*, *105*(4), 539-542.
27. Hacker, M., Jakobs, T., Hack, N., Nikolaou, K., Becker, C., Von Ziegler, F., ... & Tiling, R. (2007). Combined use of 64-slice computed tomography angiography and gated myocardial perfusion SPECT for the detection of functionally relevant coronary artery stenoses. *Nuklearmedizin*, *46*(01), 29-35.
28. Noble, J., Bourassa, M. G., Petitclerc, R., & Dyrda, I. (1976). Myocardial bridging and milking effect of the left anterior descending coronary artery: normal variant or obstruction?. *American Journal of Cardiology*, *37*(7), 993-999.
29. Greenspan, M., Iskandrian, A. S., Catherwood, E., Kimbiris, D., Bemis, C. E., & Segal, B. L. (1980). Myocardial bridging of the left anterior descending artery: Evaluation using exercise thallium-201 myocardial scintigraphy. *Catheterization and cardiovascular diagnosis*, *6*(2), 173-180.
30. Rivitz, S. M., & Yasuda, T. (1992). Predictive value of dipyridamole thallium imaging in a patient with myocardial bridging but without fixed obstructive coronary artery disease. *Journal of nuclear medicine: official publication, Society of Nuclear Medicine*, *33*(10), 1905-1913.
31. Kun, Tang, M.M., Ling, Wang, M.M., Rongfang, Shi, B.M., Xiangwu, Zheng, B.M., Ting, Li, B.M., Xiaobin, Zhao, M.M., & Ruming, L.U., BMc.(2011). The role of myocardial perfusion imaging in evaluating patients with myocardial bridging (J Nucl Cardiol, 18:117–22.)
32. Rafał, G., Jacek, K.K., Anna, Płachcin,´S.K.A. (2011). Małgorzata Bien´kiewicz, Jarosław Dro_zd, Grzegorz Piotrowski, and Krzysztof Chi_zyn´ski, Myocardial perfusion GSPECT imaging in patients with myocardial bridging (J Nucl Cardiol, 18:1059–65.)