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Hidden in the Chest: Myocardial Bridging and Cardiovascular Response to Exercise

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ABSTRACT

Myocardial bridging (MB) of the coronary arteries has long been considered a benign anatomical variant with no significant clinical relevance. However, the development of modern imaging and functional diagnostic methods indicates that its impact on myocardial perfusion may become apparent under conditions of increased hemodynamic load, especially during physical exertion. In the context of sports science, this issue is particularly important, as even subtle coronary flow disturbances can modulate the heart's response to training load and affect exercise tolerance.

The aim of this study was to present the current state of knowledge on the anatomy, pathophysiology, clinical picture, diagnosis, and therapeutic management of myocardial bridges, with particular emphasis on their functional significance under conditions of stress typical of physical activity. The article is a narrative review of the literature based on a critical analysis of anatomical, observational, and imaging studies, as well as works devoted to functional assessment and treatment strategies.

Available data indicate that MBs are a common structure, but heterogeneous in terms of morphology and hemodynamic significance. Although they remain asymptomatic in most

people, in selected cases they may be associated with myocardial ischemia, arrhythmias, and reduced exercise tolerance. Myocardial bridges require individualized clinical and functional assessment, and their presence should not be automatically considered insignificant, especially in the context of exercise capacity assessment.
Keywords: myocardial bridging, myocardial ischemia, systolic compression, coronary artery disease

INTRODUCTION

Myocardial bridging over coronary vessels (MB) is one of the issues that has been marginalized and underestimated for many years. The first records of the existence of these structures appeared in 1737, when Reyman described them, and the first detailed autopsy report containing a description of myocardial bridges created by Geiringer was not published until 1951. For decades, they were treated as an anatomical variant of the norm, devoid of hemodynamic consequences, and their presence had no impact on diagnostic and therapeutic decisions. At that time, the prevailing view focused on atherosclerotic changes as the almost exclusive cause of myocardial ischemia [1][2].

However, technological advances in our time have led to a significant revision of this approach. The introduction of high-resolution imaging techniques, such as coronary computed tomography angiography and intravascular ultrasound, together with classic coronary angiography, has enabled precise assessment of the anatomy of MB and their relationship with the surrounding heart muscle tissue [3][4]. At the same time, subsequent developments in functional assessment methods have revealed that in a selected group of patients, MB can lead to significant coronary perfusion disorders, especially in conditions of increased metabolic demand or concomitant microcirculatory dysfunction [5][6].

ANATOMY AND EPIDEMIOLOGY

By definition, a myocardial bridge is an anatomical anomaly consisting of a section of the coronary artery running inside the myocardial layer instead of its classic epicardial location. The tunneled fragment of the vessel is then referred to as an intramural segment, subject to systolic compression. From an embryological point of view, there are two theories regarding

the formation of bridges. The first refers to the primary intramural course of the artery, and the second to the bridge as a secondary structure formed by the migration of myocytes over the epicardial vessel [2][7].

Most often, bridges affect the anterior interventricular branch of the left coronary artery, especially its middle section, as proven by both autopsy studies and modern analyses based on imaging studies [8]. Less common locations of MBs include the diagonal branches, the circumflex branch (LCx), or the right coronary artery. It should be noted that these cases are mainly described in autopsy studies and are generally of limited clinical significance [7][9].

From an anatomical point of view, bridges are characterized by considerable heterogeneity, as their length can vary from a few to over 80 mm. The literature distinguishes between superficial bridges, surrounded by a thin layer of muscle fibers, and deep bridges, surrounded by a thick layer of muscle, which is associated with a significantly higher risk of coronary artery lumen compression [4].

The prevalence of MB depends on the diagnostic method used. A similar frequency is observed in both sexes, although some researchers indicate that women are at greater risk of having this structure. Autopsy studies confirm the presence of bridges in more than half of the population, while classic coronary angiography reveals them in a much more limited range, at most in a few percent of the population, but this rate may increase to as much as 40% after provocative tests. New studies using coronary computed tomography angiography show similar detectability to autopsy studies [3][8][10].

It is increasingly emphasized that the mere presence of a myocardial bridge should not be assessed binarily, but comprehensively, taking into account its length, depth, location, and spatial relationship with adjacent myocardial structures [5][8][11].

PATHOPHYSIOLOGY

The pathophysiology of myocardial bridging is primarily due to the presence of a segment of the coronary artery running intramuscularly, which undergoes dynamic compression during myocardial contraction, the degree of which depends on the anatomical features of the bridge. In most cases, this phenomenon does not lead to significant clinical consequences, but in some patients it may be hemodynamically significant [2][7].

Although myocardial perfusion occurs mainly during diastole, numerous studies have shown that in patients with pronounced bridges, systolic compression of the vessel may also partially persist in the early phase of diastole. This delays the full opening of the artery lumen and

shortens the effective coronary flow time, leading under certain conditions to a reduction in coronary reserve and a mismatch between blood supply and myocardial demand [4][12].

An important pathophysiological element is the hemodynamic changes occurring in the proximal section of the vessel relative to the MB. In this location, a different nature of blood flow is observed, which results in a reduction in the shear forces acting on the vascular endothelium. This results in increased expression of VCAM-1 molecules and the formation of reactive oxygen species, as well as the development of an atherogenic phenotype of endothelial cells. All this leads to the development of atherosclerosis in the proximal section of the vessel, which has been reflected in multiple histopathological and imaging studies, while the tunneled segment itself usually remains free of significant atherosclerotic changes [4][13].

In addition, we can distinguish several pathophysiological mechanisms that can cause symptoms of myocardial ischemia in people with muscle bridges who previously showed no symptoms:

1. Left ventricular hypertrophy - increased vessel compression and reduced microvascular reserve
2. Left ventricular diastolic dysfunction, hypertension - in combination with the presence of a MB, disturbance of myocardial oxygen supply and demand
3. Coronary vasospasm, endothelial and microvascular dysfunction, other cardiovascular factors – in combination with MB, may result in myocardial ischemia [14][15][16].

In summary, the pathophysiology of myocardial bridging is multidimensional and should not be limited to a simple model of systolic arterial compression. It is a complex spectrum of anatomical, hemodynamic, and functional disorders, the clinical significance of which translates individually to each patient.

CLINICAL PRESENTATION

The clinical presentation of myocardial bridges is varied and, in most cases, asymptomatic. In a significant proportion of patients, myocardial bridges are merely an anatomical variant, detected incidentally in imaging studies or autopsies, with no discernible clinical consequences [2]. Symptoms affect a minority of patients and most often include complaints typical of myocardial ischemia, such as chest pain, retrosternal discomfort, or exertional dyspnea, often in the absence of significant atherosclerotic changes in the coronary arteries [17][18]. Resting ECG is usually normal, and clinical symptoms associated with MB are usually exertional or occur in situations of increased myocardial oxygen demand, such as tachycardia or physical exertion. In some patients, the symptoms may be quite nonspecific and take the form of angina

“masks,” such as fatigue or abdominal pain accompanied by nausea. The poor correlation between the above-mentioned symptoms and the angiographic picture makes it very difficult to make an unambiguous clinical assessment [4][19].

Symptoms such as the following are much less common:

- acute coronary syndrome,
- ventricular septal rupture,
- cardiac arrhythmias: ventricular or supraventricular tachycardia, atrioventricular block,
- cardiac stunning,
- syncope,
- sudden cardiac death.

Unfortunately, the exact risk of these complications correlated with the occurrence of the anomaly that is the subject of our study is unknown, and in most reports they are only observational in nature [20][21][22][23].

In some patients, therefore, the correct interpretation of the clinical picture requires moving away from a purely anatomical approach and taking into account the broader clinical context and the results of functional tests, which allow for a better determination of their actual significance.

DIAGNOSTICS

The diagnosis of bridging requires a multi-stage approach combining anatomical assessment with functional analysis, because, as mentioned earlier, the presence of bridging alone does not determine its clinical significance. In most patients, MB is an incidental finding, and the real diagnostic challenge remains the identification of those patients in whom this structure can have significant hemodynamic implications [8].

Coronary angiography, a classic invasive examination, remains the historically most commonly used method for diagnosing MB. A characteristic angiographic finding is systolic narrowing of the coronary artery lumen with its re-dilatation in the diastolic phase, known as the “milking effect”. The second element that can be visualized in angiography, indicating the intramural course of the anterior descending branch, is a U-shaped morphology, best seen in lateral projections. The stenosis resulting from systolic compression can be enhanced by endovascular administration of nitroglycerin. This drug dilates adjacent vessels and non-bridged segments of coronary arteries, allowing for easier identification of MB. However, it should be remembered that the sensitivity of coronarography in detecting bridging is limited, and the degree of

compression assessed visually does not always correlate with the actual functional significance of the finding [18][24][25].

Coronary angiography can be supplemented by intravascular ultrasound (IVUS), which is usually performed during angiography by inserting additional catheters and an ultrasound camera. IVUS allows for accurate assessment of the vessel lumen dimensions and dynamic compression of the intramural segment, as well as identification of the characteristic hypoechoic crescent-shaped image of the muscle surrounding the coronary vessel. This method also allows for the assessment of the vessel wall in the proximal segment and confirmation of a greater predisposition to the development of atherosclerotic plaque in this location [12]. Due to its invasive nature, IVUS is mainly used in selected clinical cases and scientific research [25].

Invasive functional measurements within myocardial bridges, especially during pharmacological provocation, can provide a lot of valuable information regarding their clinical significance. They allow for the assessment of the constant component of stenosis, but also the dynamic compression of the vessel, which may be responsible for the occurrence of ischemia symptoms, as well as the identification of coexisting endothelial dysfunction or coronary vasospasm.

The bridged segment in intravascular Doppler measurements is characterized by a typical flow profile with rapid acceleration in the early diastolic phase (the “finger tip” phenomenon), resulting from a simultaneous decrease in microcirculatory resistance and persistent muscle compression, followed by a rapid deceleration and a plateau phase after full opening of the vessel lumen [12].

Functional assessment uses fractional flow reserve (FFR) measurement, with values <0.75 suggesting ischemia and the range 0.75-0.80 remaining a “gray area” where even a dobutamine test does not rule out the possibility of false negative results. In patients with an FFR >0.80, the administration of dobutamine may lead to the recurrence of stenocardiac symptoms and suggest the significance of the MB. In order to exercise diagnostic caution, an iFR (Instantaneous Wave-Free Ratio) test can be performed, but it should not be forgotten that in the myocardial bridge, impaired flow also occurs during diastole [26][27].

Coronary CT Angiography is currently the primary tool for non-invasive diagnosis of myocardial bridges. This method allows for precise visualization of the artery course, assessment of the length of the tunneled segment, its depth within the myocardium, and classification as a superficial or deep bridge. The test is highly sensitive in detecting MB and

often reveals bridges that are invisible in coronary angiography, but, like coronary angiography, it still does not allow for an unambiguous assessment of their hemodynamic significance, although research on the development of this method is ongoing [3][28].

Non-invasive diagnostics are supplemented by methods that assess myocardial perfusion and contractility. SPECT allows the detection of reversible perfusion disorders in patients with myocardial bridges, and the severity of ischemic changes may correlate with the degree of systolic narrowing of the coronary artery lumen. The echocardiographic dobutamine test has similar diagnostic value, in which transient contractility disorders are observed in areas supplied by the bridged artery [19][29][30].

A comprehensive assessment of the clinical significance of MB should therefore take into account both the anatomical picture and the results of functional tests, which, when combined, provide a starting point for the appropriate selection of a therapeutic strategy.

TREATMENT

Advances in diagnostics have led to myocardial bridges being detected in an increasing number of people. With this in mind, it is important to note that the implementation of therapy should depend on the presence and severity of clinical symptoms and the hemodynamic significance of the change. In the vast majority of patients, MB is only an anatomical finding and treatment is not necessary, but the approach is completely different in symptomatic patients [2].

The first-line treatment is pharmacotherapy, which aims to reduce the heart rate, prolong diastole, and limit the force of myocardial contraction. The best-documented group of drugs are β-blockers without vasodilatory effects, which, through the above-mentioned actions, improve coronary perfusion during diastole and reduce stenotic symptoms. An alternative in patients with contraindications to β-blockers or intolerance to this group of drugs are calcium channel antagonists, especially in cases of suspected concomitant coronary vasospasm [18].

The use of nitrates in the treatment of myocardial bridging is not recommended because they cause a decrease in vascular wall tension, and the artery then becomes more susceptible to compression by the surrounding muscle, which may lead to worsening ischemic symptoms [24].

In patients with persistent symptoms despite optimal pharmacotherapy, invasive treatment should be considered, which can be divided into percutaneous interventions using stents and surgical treatment.

Currently, patients suffering from severe angina symptoms that do not subside despite the aforementioned pharmacotherapy undergo surgical myotomy, where a cardiac surgeon cuts the heart muscle fibers that run over the artery, thus releasing it from compression. Potential

complications that may affect the patient after surgery include postoperative hemorrhage, vessel perforation, or ventricular aneurysm.

Another surgical method that can be used is classic coronary artery bypass grafting (CABG), most often using the left internal mammary artery. This method involves performing a vascular anastomosis that bypasses the bridged segment of the vessel, allowing for a reduction in ischemic symptoms in the patient [31][32].

Unfortunately, there are no studies that would clearly assess which of these two procedures is better. In the case of deep and long bridges, CABG is preferred. On the other hand, myotomy, as a procedure that repairs the underlying pathology, is the treatment of choice in symptomatic patients who do not respond to pharmacological treatment, with symptoms of ischemia or significant systolic compression visible in imaging studies [33][34].

The last invasive treatment method used is percutaneous coronary intervention (PCI). These procedures have many advantages, namely they avoid surgical treatment and improve coronary flow. Unfortunately, they have a significant disadvantage, which is the high risk of restenosis, i.e., re-narrowing of the vessel lumen at the stent implantation site [35]. Patients who underwent percutaneous procedures required revascularization much more often, which significantly limits the use of this method, so the choice of treatment strategy requires individual clinical and functional assessment of the patient.

CONCLUSION

Myocardial bridging of the coronary arteries is an example of an anatomical structure whose clinical significance remained unknown for a long time. As demonstrated in this study, current data clearly indicate that MB is not a homogeneous and clinically neutral phenomenon, and its impact on myocardial perfusion depends on many coexisting factors. The morphological characteristics of the bridge, coronary flow dynamics, the presence of microcirculatory disorders, and conditions of increased metabolic demand, including physical exertion, play a key role.

Advances in imaging and functional assessment techniques have made it possible to move away from a purely anatomical view of MBs in favor of a more precise, individualized assessment of their hemodynamic significance. Of particular importance is the synthesis of data obtained from non-invasive and invasive studies, which allows for the proper qualification of patients for observation, pharmacological treatment, or invasive therapy. In this context, myocardial bridges should be considered not as an isolated anomaly, but as a potential element of the complex pathophysiology of myocardial ischemia.

From a clinical point of view, the most important challenge remains the identification of a small group of patients in whom MB has real symptomatic and prognostic significance. Rational management requires a balance between avoiding overdiagnosis and unnecessary interventions, and the correct diagnosis of patients at risk of ischemic complications. Only such a comprehensive and evidence-based approach allows for the safe and effective management of patients with myocardial bridging in everyday medical practice.

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Author's contribution:

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