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Міокардіальні містки у поєднанні з феноменом WPW: огляд літератури та клінічний випадок

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Природжена аномалія коронарних судин у вигляді міокардіальних м'язових містків (ММ) найчастіше локалізується в системі лівої коронарної артерії, у середньому сегменті передньої міжшлуночкової гілки. Звичайно вважається доброякісним станом, може перебігати асимптомно. Але встановлені зв'язки наявності ММ з низкою клінічних проявів, деякі з яких є загрозливими для життя та здоров'я пацієнтів, не вирішеними до кінця питаннями лікарської тактики ведення таких осіб, особливостями надання допомоги при ускладненнях, визначенням потреби втручання за умов асимптомного перебігу. В статті надані сучасні погляди на особливості клініки, діагностики, лікування симптомних пацієнтів з ММ, з врахуванням супутньої ішемії міокарда. Описаний

клінічний випадок характеризується слабо вираженим у пацієнта абдомінальним больовим синдромом після фізичного навантаження, поєднанням наявності міокардіального містка в лівій коронарній артерії, з високим ступенем систолічного стискання судини, розповсюдженою субендокардіальною ішемією, асоційованою з фізичним навантаженням, та зафіксованим у пацієнта феноменом WPW.

Ключові слова: міокардіальні м'язові містки, аномалії коронарних артерій, феномен WPW.

Myocardial bridging and WPW phenomenon: Case-based review

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A congenital anomaly of coronary vessels in the form of myocardial muscle bridges (MBs) is most commonly located in the left coronary artery's system, specifically in the middle segment of the anterior interventricular branch. Typically considered a benign condition, it can be asymptomatic. However, the presence of MBs has been associated with various clinical manifestations, some of which pose threats to the life and health of patients, unresolved issues that include medical management tactics for handling such patients, specifics of assisting during complications and determining the need for intervention in asymptomatic cases. This article presents contemporary perspectives on the clinic, diagnosis, and treatment of symptomatic patients with MBs, considering concomitant myocardial ischemia. We presented a case of a 41-year-old male with the myocardial bridge in the left coronary artery characterized by a high degree of systolic compression of the vessel, widespread exercise-induced subendocardial ischemia with abdominal pain and WPW phenomenon.

Keywords: Myocardial muscle bridges, anomalies of coronary vessels, WPW phenomenon.

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Introduction

Recently, the issue of myocardial muscle bridges (MBs) in coronary arteries has garnered particular attention [1, 2, 3]. This anomaly is characterized by the intramural passage of a vessel segment, "immersing" beneath the myocardium. Connections have been established between the presence of MBs and several clinical manifestations, some of which pose threats to the life and health of patients. Angiographically, MBs are visualized in 0.5–16% of people [4, 5, 6], while during autopsies, such "tunnel" coronary arteries are found in 40–80% of cases [4]. According to other data, the prevalence ranges from 5 to 86%, with an average of 25% during autopsies [5, 6].

MBs in a patient can be considered a benign condition with a favorable long-term prognosis. It often does not manifest itself clinically and can be detected accidentally during coronary angiography or autopsy [7]. The hemodynamic influence of MBs depends on the thickness, length, and location relative to the myocardium fibers and surrounding tissue [4]. Clinical manifestations may occur with increased load on the heart, especially in young people; this is facilitated by hypertrophy and fibrosis of the myocardium of the left ventricle [4; 6]. Evaluating anatomical characteristics helps in predicting the clinic and its consequences. However, issues regarding the medical management tactics for handling such patients remain unresolved, as well as specifics of assisting during complications and determining the need for interventions in asymptomatic cases.

We performed a literature search through Scopus and PubMed, analyzing research articles related to MBs and presenting our clinical case.

Case description

In January 2019, Patient M, a 41-year-old lawyer, presented for a routine medical examination. He reported occasional epigastric discomfort and palpitations, particularly after physical exertion, noted over the previous year.

Body Mass Index (BMI) was 30 kg/sq. m, indicating obesity. The examination revealed no pathological changes; his skin was clear, lymph nodes were not enlarged, and there was no edema. Respiratory and cardiovascular

assessments were normal, with a heart rate of 54 beats per minute and blood pressure at 125/75 mm Hg. The gastrointestinal examination showed a soft, non-tender abdomen with no palpable spleen and the liver palpable near the costal margin.

Upon laboratory examination, the complete blood count was normal. The blood biochemical analysis showed the following results: serum glucose – 5.07 mmol/L, total cholesterol – 4.8 mmol/L, HDL-cholesterol – 1.25 mmol/L, LDL-cholesterol – 3.20 mmol/L, and triglycerides – 1.6 mmol/L.

Esophagogastroduodenoscopy: no gastrointestinal pathologies; negative for *Helicobacter pylori*. Ultrasound: normal findings in abdominal organs and thyroid gland.

Electrocardiography (ECG) showed regular sinus rhythm – 53 beats per minute, PR – 135 ms, P wave – 117 ms, QRS – 107 ms, T wave – 235 ms, and QT/QTc – 425/402 ms. The alpha angle was +1°, and the electric axis was horizontally positioned. A delta wave was observed in the leads V2-V4 on the ascending part of the QRS complex.

Considering identifying signs of pre-excitation syndrome and complaints of interruptions in the heart's work, the patient was referred for advanced cardiovascular testing such as bicycle ergometry (BE), 24-hour N.J. Holter ECG monitoring (HM), and heart ultrasound.

The BE results were: heart rate before the test (P) – 60/min, and blood pressure (AP) – 140/85 mm Hg.

1st stage of loading: workload 50 W, P – 110/min, BP – 180/90 mm Hg.

Recovery: 1 min – P – 80/min, BP – 175/80 mm Hg; 3 min – P – 70/min, BP – 150/80 mm Hg; 6 min – P – 80/min, BP – 138/75 mm Hg; 10 min – P – 70/min, BP – 120/80 mm Hg; 15 min – P – 70/min, BP – 120/75 mm Hg. The test was terminated after the 1st stage of loading.

Conclusion. The test result is positive. During the first loading stage, subendocardial ischemia of the anterior-septal-apical-lateral wall of the left ventricle was detected against the

background of sinus tachycardia. The cardiovascular system's response was indicative of both ischemic and hypertensive types. Recovery was slow. The patient was observed at rest for 22 minutes until repolarization processes resumed. No signs of subendocardial ischemia were registered, and the heart rate stabilized at 65–70 beats per minute. A single right ventricular extrasystole was recorded throughout the entire BE test during the 1st minute of loading. The Wolff-Parkinson-White (WPW) phenomenon persists.

Results of HM: ECG features: A delta wave was observed, and PR was shortened to 100 ms, suggesting a probable phenomenon of WPW. Holter ECG monitoring lasted one day and 5 minutes, including 23 hours and 59 minutes suitable for analysis. Recording was conducted in modified bipolar leads V1M, Y, and V5M. The average daily heart rate (HR) was 55 beats per minute, averaging 62 beats per minute during the day and 46 beats per minute at night; the minimum HR was 36 beats per minute, and the maximum HR was 127 beats per minute (circadian index 135% that is within the normal range). Bradycardia was observed during the day. Against the background of sinus rhythm, the following arrhythmias were detected:

- a single monomorphic ventricular extrasystole, including early R-on-T types, totaling 503 extrasystoles (above average value), as well as an increase in extrasystoles during physical loading;
- a single atrial extrasystole, totaling 5 (within the normal range). No clinically significant pauses were registered.

Submaximal heart rate was not achieved during physical loading (stair tests). Tachycardia-dependent oblique ascending and horizontal depression of the ST segment in the leads Y and V5M, with a maximum depression of up to 1.5 mm, was observed. Tolerance to loading was at an above-average level. The daily dynamics of the QT interval duration were within the normal range.

Heart ultrasound revealed the following measurements: right ventricle – 2.4 cm, interventricular septum – 1.1 cm, left ventricle – 4.5 cm, left ventricular wall during diastole – 1.1 cm, left atrium – 4.1 cm, ascending aorta diameter – 3.1 cm, and ejection fraction – 65%.

Heart valves operated normally, and there was no evidence of pulmonary hypertension or fluid in the pericardial cavity. Segmental contractility was undisturbed, and there were no diastolic disorders. Conclusion: initial left atrium dilatation was noted, but the dimensions of other heart chambers were within normal limits. The structure and function of the valves showed no abnormalities. The contractility of the left ventricle was unimpaired.

The differential diagnosis primarily focused on distinguishing between gastrointestinal and cardiac etiologies of the patient's symptoms, leading to the identification of significant coronary artery involvement. While examining the cardiovascular system, the pre-excitation phenomenon of the Wolff-Parkinson-White (WPW) type was noted. During BE, subendocardial ischemia of the anterior-septal-apical-lateral wall of the left ventricle occurred in the background of sinus tachycardia. The cardiovascular system's response was indicative of both ischemic and hypertensive types with slow recovery. N.J. Holter's 24-hour ECG monitoring revealed multiple ventricular extrasystoles, including early ones. These extrasystoles, along with the oblique ascending and horizontal depression of the ST segment, were associated with physical loading. Structural and functional disorders, except for the initial dilatation of the left atrium, were not detected during the heart ultrasound. Based on these results, the patient was referred to the interventional radiology department for coronary angiography.

Coronary angiography findings. The examination revealed a right-type anomaly, with a muscular bridge in the medial segment of the anterior interventricular branch of the left coronary artery, causing stenosis up to 95% during systole. The right coronary artery showed no narrowing (Fig.1, Fig. 2).

A differential diagnosis between gastrointestinal pathology and cardiac diseases was performed. An anomaly in the left coronary artery system, MBs, was identified, and no pathology of digestive organs was detected.

The patient was recommended lifestyle modification for obesity to reduce lipid levels and surgical treatment of the Wolff-Parkinson-White phenomenon.

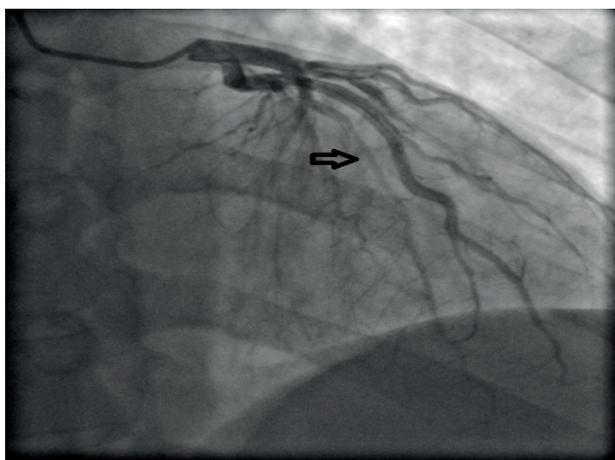


Figure 1. Coronary angiography: Stenosis up to 95% during systole

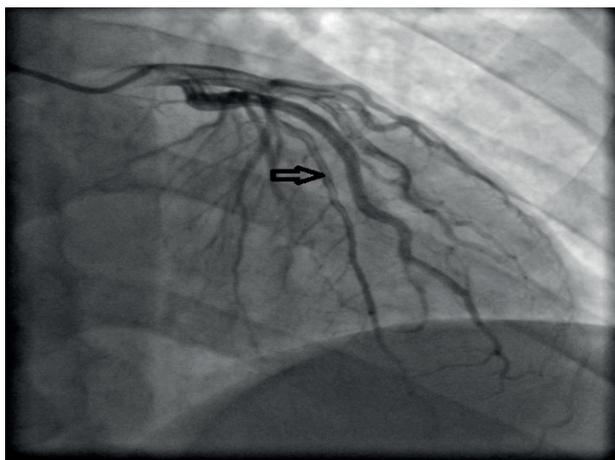


Figure 2. Coronary angiography: A muscular bridge in the medial segment of the anterior interventricular branch of the left coronary artery

Discussion

Myocardial MBs in the left descending artery system of the patient were detected. The most common localization of MBs occurs in the left coronary artery (LAD – left anterior descending coronary artery), particularly in the middle segment of the anterior interventricular branch [1]. According to Saito Y et al., 2017, MBs were predominantly located in the middle segment of the LAD system, accounting for 36% of cases. Patients with this localization often exhibited a higher frequency of provoked vasospasm and a positive acetylcholine test [8]. MBs were less prevalent in the right coronary artery system, and in cases where two parallel branches existed, one

was frequently situated intramurally. Systolic compression and narrowing of a coronary artery segment led to reduced or decreased antegrade blood flow. However, changes were detected during diastole while the vessel fully or partially expanded. It is important to note that MBs in coronary arteries can be considered an anatomical variant rather than a congenital anomaly [9].

The typical depth of MBs ranged from 1 to 10 mm with a length of 10 to 30 mm. M surfaces of ≥ 2 mm were detected depending on the integumentary muscle layer. During diastole, the diameter of the intramural segment was 34–51% smaller than the proximal segment, and the intima was thinner [5]. Hypoplasia of the “tunnel” artery intima was recorded with a width of approximately $66 \mu\text{m}$ beneath the MBs, in contrast to the intima, which reached nearly $406.6 \mu\text{m}$ before MBs [10]. Although the development of myocardial infarction under conditions of a combination of MBs and coronary arteries without obstruction is a relatively common condition, the relationship between them has not been sufficiently studied [11]. Systolic compression of the vessel contributes to damage and dysfunction of the endothelium, especially under conditions of tachycardia with diastolic narrowing. This impairment of diastolic filling and increased compression affects myocardial perfusion [4]. Mechanical desynchronies during systole may contribute to the progression of endothelial dysfunction associated with the development of atherosclerosis [12]. The degree of coronary artery obstruction is influenced by its location, the length and thickness of MBs, and the contractility of the myocardium. More common and deep MBs are associated with high systolic compression and pose a significant risk factor for coronary artery spasm [13]. According to Saito Y et al., 2017, MBs were detected in 36% of cases in the LAD system, mainly in the middle segment. Such patients exhibited a higher number of provoked vasospasms, and their acetylcholine tests were positive [8]. Systolic compression and coronary artery segment narrowing led to a decrease or even cessation of antegrade blood flow. However, changes were detected during diastole while the vessel was fully or partially expanded. The length of MBs was positively correlated with the degree of systolic compression, and these

percentage values significantly predicted the provoked spasm in the LAD [8]. The study of the clinical significance of this phenomenon continues, given that the vast majority of blood flow in the left coronary artery system occurs during diastole – ranging from 2/3 to 85% as per various data. Assessing the influence of MBs in the LAD on the perfusion of the entire myocardium of the left ventricle revealed a connection with impairment of perfusion reserve and an indicator of microvascular dysfunction. However, there was no connection with anatomical characteristics [14]. A 5-year observation of patients with MBs and a coronary spasm clinic established a high recurrence rate; intensive drug therapy and careful clinical observation of patients are necessary for better clinical results [13].

For the “tunnel” segment of the coronary artery, unlike the proximal one, the development of atherosclerosis is not typical. A lower calcium content was found in the intramural segment of the coronary artery compared to the proximal one [15]. It has been suggested that the absence of atherosclerotic changes is a consequence of the local perivascular adipose tissue, which produces pro-inflammatory adipokines and cytokines, not affecting the tunnel part of the vessel [15]. Hemodynamically significant MBs are associated with increased fibrosis and interstitial edema. In individuals who died suddenly and had no other cause of death, the electric instability of the myocardium was the probable cause. Quantitative determination of fibrosis allows for predicting its arrhythmogenic effects [16]. Under conditions of MB presence, deterioration of cardioregulatory indicators was determined; autonomic dysfunction was probably caused by recurrent myocardial ischemia [17]. Stenosis of the coronary artery containing MBs, as well as the length of the “tunnel” segment, was associated with the prevalence of systolic desynchrony, especially in patients with hypertension and more pronounced vessel overlap, regardless of left ventricular hypertrophy [18].

Manifestations of angina pectoris, myocardial infarction, left ventricular dysfunction, and ventricular tachycardia are observed in patients with MBs; sometimes, they lead to sudden cardiac death following physical activity

[3]. Acute coronary syndrome (ACS) is characterized by the disturbance of coronary blood flow, subsequent platelet aggregation, vaso-spasm, and thrombosis [19, 20]. Coronary artery spasm is the cause of both rest and exertional angina pectoris. Systolic compression of MBs can potentially provoke the rupture of plaques in the segment proximal to MBs, explaining MB-related ACS, particularly myocardial infarction in young individuals [21]. Analysis of numerous scientific works suggests the potential significance of MBs and a possible correlation with major cardiac events. However, it emphasizes the need for additional studies to confirm or refute the relationship with myocardial infarction, sudden death, and other cardiac pathologies, including Takatsubo cardiomyopathy [22, 3]. The authors note a high degree of variability in results associated with a limited number of studies, many lacking comprehensive inclusion and exclusion criteria, a complete data description, and different methodologies.

The presence of MBs has been identified in patients with hypertrophic cardiomyopathy [23]. An association with acute allograft failure during heart transplantation has also been recorded [7]. In young patients, MBs are a common cause of chest pain, and their prevalence is significantly higher in patients without severe atherosclerotic coronary stenosis with less common occurrences of hyperlipidemia [24]. Among young athletes, MBs rank as the second leading cause of death after hypertrophic cardiomyopathy [20]. It is recommended that young and professional athletes undergo echocardiography to detect hidden heart diseases, identify risk factors, and assess troponin levels [25]. Painless ischemia or ST segment depression during stress testing of athletes may indicate the need for coronary computed tomography, advanced diagnosis, and risk stratification [20]. Our patient has widespread subendocardial ischemia during physical activity associated with a high degree of systolic compression of the vessel. Although the presence of MBs is generally considered a benign condition, exercise-related cardiac events can occur. However, there is currently no evidence to suggest that asymptomatic individuals without clinical signs of ischemia should be restricted from engaging in active activities [26].

The diagnosis of MBs shall be conducted using various modern methods. Coronary angiography reveals characteristic phenomena, with the main one being the "milking effect" (systolic squeezing of contrast during the narrowing of the "tunnel" segment and partial straightening of the intramural part in diastole) [27, 4, 6]. The "step down-step up" phenomenon may also be observed, indicating a phase-by-phase contrast filling of the "tunnel" artery. Single-photon emission CT (SPECT/CT) and positron emission tomography (PET/CT) shall be performed to determine the functional significance of MBs. The perfusion defect is correlated with the degree of artery narrowing during systole. The absence of clinical manifestations is associated with a change in vessel caliber of less than 50%, while clinical symptoms are linked to an increase of more than 70% [28]. Intravascular ultrasonography and dopplerography for diagnosing MBs have revealed that vessel compression persists during diastole, contributing to decreased coronary blood flow reserve [7].

Asymptomatic patients with MBs generally do not require treatment [12]. However, the combination of mechanical compression of MBs and atherosclerotic changes in the segment proximal to MBs can lead to myocardial ischemia and may be the cause of coronary spasms. The coexistence of MBs and coronary spasms has recently garnered increased interest regarding diverse treatment approaches under these conditions [29].

The muscle bridge's mechanical compression of the coronary artery is considered the underlying cause of symptoms occurring during physical activity [29]. The primary treatment for symptomatic patients involves β -blockers, which reduce heart rate, increase the diastolic filling force of coronary arteries, and reduce their contractility and compression, alleviating hemodynamic disorders [30]. However, such monotherapy can exacerbate symptoms of coronary spasm, leading to spontaneous angina and angina pectoris [29]. Nitrates, effective for coronary spasms, may worsen symptoms in patients with muscle bridges, increase systolic compression of the bridge segment, elevate retrograde blood flow in the proximal segment, and lower the ischemic threshold. Therefore, carefully considering both pathol-

ogies is necessary when determining optimal drug therapy for symptomatic patients [4, 29]. Antiplatelet drugs and blockers of slow calcium channels are prescribed in cases of concomitant vasospasm [30, 4]. Surgical treatment is typically considered when there is poor tolerance to β -blockers. Procedures such as stenting the "tunnel" segment and aorto-coronary shunting are performed. Myotomy is a priority surgical intervention for symptomatic patients, improving coronary blood flow and relieving coronary artery compression [31].

We performed a differential diagnosis between possible gastrointestinal pathology and cardiac diseases. An anomaly in the left coronary artery system in the form of MBs was identified, which explained discomfort during physical activity due to the mechanical compression of the artery by the myocardial bridge. Special attention was drawn to the observed high degree of systolic compression of the vessel, widespread subendocardial ischemia associated with physical activity, and the atypical abdominal nature of the mildly expressed pain syndrome. The patient was characterized by a congenital coronary artery anomaly in the form of MBs combined with the WPW phenomenon, posing a risk of paroxysms of supraventricular tachyarrhythmias. This combination was previously described by Sueda T et al. (1991) [32]. The recorded early ventricular extrasystole was concerning, as R-on-T type extrasystoles could trigger Torsade de Pointes ventricular tachycardia, posing a threat of transitioning to ventricular fibrillation (VF). Endothelial dysfunction caused by MBs and associated coronary angiospasm may explain cases of ischemia-induced VF [33]. Symptoms of ischemia in the background of MBs led to the adoption of various modern functional and anatomical imaging methods, significantly deepening the understanding of the dynamic pathophysiology associated with MBs [34]. β -blockers were not prescribed to the patient due to daytime bradycardia confirmed by HM data. Supervision by a cardiologist and a cardiac surgeon, conducting an electrophysiological examination, and resolving the issue of the need for catheter ablation of additional conducting pathways and operative treatment of MBs, particularly myotomy, was recommended. Considering first-degree obesity, diet therapy with the exclusion of additional physical activity was prescribed as well.

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