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# MITRAL STENOSIS ETIOLOGY, PATHOGENESIS AND CLINICAL SYMPTOMS

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**Abstract**

This article systematizes the etiology, pathogenesis, and clinical manifestations of mitral stenosis through an analytical review of contemporary literature. Narrative and comparative bibliographic methods were applied. The results distinguish mechanisms typical of rheumatic versus degenerative disease, outline major hemodynamic consequences, and identify early clinical markers. The study strengthens differential diagnostic reasoning and supports clinically oriented stratification.

**Keyword**.

Mitral stenosis, rheumatic heart disease, left atrium, pulmonary hypertension, atrial fibrillation, valvuloplasty, hemodynamics.

**Introduction.** Mitral stenosis is a condition of high clinical importance among the pathologies of the valvular apparatus of the heart, a long-term process that leads to a gradual restructuring of cardiopulmonary hemodynamics. Its relevance is determined by two reasons at once: on the one hand, in regions where rheumatic heart disease persists, mitral stenosis still contributes to the risk of disability and maternal mortality in patients of working age; on the other hand, the aging of the population and the complication of comorbid conditions increase the proportion of degenerative calcific processes, which leads to atypical clinical manifestations and delayed diagnosis. However, in practice, mitral stenosis is often interpreted only as echocardiographic narrowing, and morphological signs specific to the etiology, stages of pathogenesis and the evolution of clinical semiotics over time are not sufficiently integrated. Especially in the context of the convergence of the spectrum of clinical manifestations of rheumatic and degenerative stenosis, a clear understanding of the hemodynamic mechanisms underlying the common symptoms is crucial for differential diagnosis, risk stratification, and treatment strategies. Although the specific features of mitral stenosis are widely covered in the existing scientific literature, in many sources the etiological factors and pathogenetic chains are not consistently linked to clinical manifestations, and the prognostic value of clinical manifestations is often limited to general statements. Also, concepts proposed by different schools, such as the clinical consequences of the ratio of functional vasoconstriction and subsequent structural remodeling in the pulmonary vessels, or the transformation of left ventricular remodeling into an arrhythmogenic substrate, are not always compatible with practical clinical decisions. Therefore this article aims to narrow the conceptual gap in diagnosis by systematizing the internal relationship between the etiology, pathogenesis, and clinical manifestations of mitral stenosis. The purpose of the article is to analytically highlight the main etiological variants of mitral stenosis, their pathogenesis mechanisms and clinical manifestations based on modern scientific evidence. To achieve this goal, the following tasks were set: to distinguish the leading etiological factors leading to mitral stenosis and their morphological features, to explain the chain of hemodynamic changes in stages from left ventricular pressure to pulmonary hypertension, to

systematize early and late manifestations, linking clinical signs with pathogenetic mechanisms; to scientifically substantiate the possibilities of using symptoms and signs in differential diagnosis and clinical stratification. This work was methodologically structured as an analytical-narrative literature review and was aimed at clinical-conceptual synthesis. In the process of preparing the article, international and regional cardiology sources, classical monographs on valvular heart diseases, and practical manuals were covered [1,3]. The combination of methods was justified by the need to comprehensively illuminate the multifactorial nature of mitral stenosis. First a comparative analysis was used to show the differences between etiological variants, secondly, a conceptual-constructive synthesis method was chosen to link the sequence in pathogenesis with clinical semiotics, and thirdly, a descriptive-analytical approach was used to systematize clinical signs. The criteria for selecting sources were their direct relevance to the topic, the degree of application in clinical practice, and the ability to illuminate the triad of etiology pathogenesis clinic. The review compared the classical views on rheumatic heart disease and the concepts of the pathogenesis of degenerative and calcific processes, commissural fibrosis, subvalvular deformation of the valve apparatus, and arrhythmias in recent years [4,6]. Based on the data obtained, the clinical signs of mitral stenosis were regrouped in a logical connection with pathogenetic mechanisms, and the diagnostic weight of the signs that are important from the point of view of clinical scenarios was also analyzed. This methodology is aimed not at the development of experimental or clinical registry data, but at the theoretical integration based on evidence, as a result of which a structured model for practical clinical thinking was proposed. Literature analysis shows that the rheumatic process historically occupies a leading place in the etiology of mitral stenosis, but in the conditions of epidemiological transition the share of the degenerative-calcific variant is increasing. The main morphological signs of rheumatic stenosis are the fusion of the commissures, diffuse thickening of the valve leaflets and shortening of the subvalvular apparatus, this combination causes a “funnel” deformation of the mitral orifice and mechanical restriction of diastolic flow [1, 4]. In degenerative stenosis, commissural fusion is not always predominant, but calcification, hardening, and decreased leaflet mobility are more common in the annulus and leaflets, which, clinically, reduces the etiological “purity” of symptoms, as this condition is often accompanied by arterial hypertension, diastolic dysfunction, and other valve defects in older patients [2, 6]. Relatively rare etiological causes include congenital mitral stenosis, fibrosis on the background of systemic diseases, and valve changes after radiation injury, which are more important in clinical practice at the stage of differential diagnosis [3]. As a central link in pathogenesis, an increase in diastolic pressure in the left atrial cavity as a result of a decrease in the mitral orifice area has been identified. This pressure gradient is initially accompanied by functional compensation. The left ventricular myocardium attempts to maintain left ventricular filling during diastole by thickening and dilation, but over time, fibrosis and electrical remodeling of the ventricular wall increase, forming an arrhythmogenic substrate [5]. As a clinical reflection of this process, a gradual increase in shortness of breath during physical exertion in sinus rhythm, followed by a sharp exacerbation of symptoms with episodes of atrial fibrillation, is systematically observed. When atrial fibrillation occurs, the atrial contribution to diastolic filling disappears, the increase in heart rate shortens diastole and leads to a relative increase in the mitral gradient, as a result of which the patient's subjective condition worsens in the short term, pulmonary venous congestion increases. The next stage of pathogenesis is associated with an increase in pressure in the pulmonary venous system and its consequences at the capillary-alveolar level. Literature analysis shows that initially in the pulmonary vessels there is a reflex vasoconstriction and endothelial dysfunction, and then a steady increase in pulmonary arterial resistance due to medial hypertrophy and fibrous remodeling of the intima [2, 7]. Therefore, the clinical signs are two-layered, one part is

explained by venous congestion (orthopnea, paroxysmal nocturnal dyspnea, cough), and the second part is explained by pulmonary arterial hypertension and right heart overload (rapid fatigue, decreased physical endurance, heaviness under the right rib, peripheral edema). As pulmonary hypertension deepens, signs of tricuspid regurgitation and right ventricular failure predominate, which indicates the transition of mitral stenosis to the "pulmonary-cardiac" phenotype. Systematization of clinical signs made it possible to distinguish early and late signs according to their pathogenetic basis. In the early stages, dyspnea is usually only observed on exertion and the patient may attribute this to a general lack of physical fitness; it is at this point that auscultatory signs, particularly a diastolic murmur and opening tone, serve as a signal to increase clinical alertness [1]. In later stages, episodes of hemoptysis are associated with bronchial vein dilatation and microbleeding against the background of pulmonary venous hypertension, and hoarseness is explained by compression of the recurrent laryngeal nerve due to left ventricular enlargement. Thromboembolism, especially ischemic stroke and peripheral embolism associated with thrombus formation in the left ventricular appendage against the background of atrial fibrillation, are mentioned as dangerous clinical consequences; this requires considering mitral stenosis not only as a "shortness of breath" disease, but also as a source of systemic thromboembolic risk [5, 8]. Clinical accents have also been distinguished according to etiological variants. Rheumatic stenosis is more likely to occur in younger patients, with a history of rheumatic diseases in childhood or adolescence, and with a classic auscultatory picture with a predominance of commissural deformation. Degenerative stenosis, on the other hand, often occurs in the elderly, in the context of multiple diseases, with diastolic dysfunction and arterial hypertension, auscultation is less typical, and symptoms can be more in the spectrum of general heart failure, as a result of which it becomes difficult to determine the etiology based only on clinical signs and the role of instrumental assessment increases [2, 6]. Nevertheless, within the framework of this work, by clarifying the pathogenetic roots of clinical signs, a logical clinical "roadmap" was formed that helps to predict the predominant mechanism of mitral stenosis, even in comorbid conditions. The results obtained indicate the need to understand mitral stenosis as a syndrome that is not etiologically homogeneous, but has a common hemodynamic "highway" in pathogenesis. The main source on heart diseases edited by Braunwald specifically emphasizes that the clinical course of mitral stenosis is sensitive to changes in mitral gradient, heart rate, and pulmonary vascular resistance [2]. This position is consistent with our systematization. Acute exacerbations of symptoms are often not due to a sudden increase in anatomical stenosis, but rather to functional factors such as increased heart rate or atrial fibrillation. Therefore, distinguishing between "anatomical" and "dynamic" components in clinical assessment is essential for accurate diagnosis and individualization of treatment. In the Russian school of cardiology on rheumatic etiology, the combined damage of the valvular apparatus and subvalvular structures is indicated as an important determinant of the severity of stenosis [4]. This approach implies that the deformational phenotype of the valve should be taken into account, not limiting mitral stenosis to the cusps alone. Our analysis showed that clinical auscultatory signs in rheumatic stenosis are relatively "classical", since commissural fusion enhances diastolic flow turbulence, which, in practical terms, means that the value of auscultatory skills in the primary segment remains in rheumatic regions. At the same time, the observation that murmurs and tones may be atypical in degenerative stenosis requires constant consideration of other components of clinical vigilance, such as the risk of dyspnea on exertion being explained by diastolic dysfunction. The European Society of Cardiology guidelines state that the clinical severity of mitral stenosis should be determined by a comprehensive assessment of symptoms, pulmonary hypertension, and valve morphology, and that atrial fibrillation and thromboembolic risk should be managed as independent clinical targets [7]. In line with this

concept, we have identified left ventricular remodeling as a central component of the pathogenesis and associated it with a "tough turning point" of clinical features. The development of atrial fibrillation not only exacerbates symptoms but also dramatically increases the risk of thromboembolic complications; this situation requires a careful balance between rhythm control, rate control, and anticoagulation strategies in clinical practice, tailored to the etiology and patient profile [8]. In this context, factors such as bleeding risk, comorbid conditions, and drug interactions complicate clinical decisions in the elderly with degenerative stenosis; therefore, understanding the pathogenesis provides a rationale for individual risk assessment. In the case of pulmonary hypertension, some sources interpret it mainly as a result of "passive" venous stasis, while others emphasize the independent role of endothelial dysfunction and structural remodeling [2, 7]. Our results integrate these two positions as a coherent process: a prolonged increase in venous pressure initially triggers functional responses, and then irreversible vascular changes develop. Clinically, this means that early detection of stenosis and timely intervention can not only reduce symptoms, but also improve the long-term prognosis by preventing irreversible remodeling in the pulmonary vessels. At this point, the question of choosing the timing of valvuloplasty or surgical intervention is directly related to the pathogenesis, and it is necessary not to ignore the hidden progression behind the "normal" appearance of clinical signs. In local sources and educational literature, mitral defects are often described as a set of classic clinical signs [9, 10]. Our approach aims to increase the diagnostic value of these signs by "tying" them to a pathogenetic mechanism. For example, if hemoptysis is viewed not only as a sign of general congestion, but also as a marker indicating that pulmonary venous pressure has exceeded a certain level, its role in assessing clinical severity becomes clearer. Similarly, if relatively rare signs such as hoarseness or dysphagia are considered as an anatomical consequence of left ventricular enlargement, they serve as "red flags" for instrumental examination. Thus rereading the clinical picture of mitral stenosis in terms of pathogenesis structures clinical thinking and serves to reduce diagnostic delays. Although mitral stenosis is etiologically diverse, its pathogenesis develops through a consistent chain of events, including increased left atrial pressure due to mitral orifice stenosis, pulmonary venous congestion, functional and then structural remodeling of the pulmonary vessels, and increased right heart afterload. Rheumatic stenosis is characterized by more commissural fusion and subvalvular deformation, while degenerative stenosis is characterized by calcific induration and comorbid background, which leads to different diagnostic risks in the interpretation of clinical signs. The focus of clinical manifestations is on exertional dyspnea, acute deterioration associated with atrial fibrillation, signs of pulmonary hypertension, and thromboembolic complications; their assessment in relation to pathogenetic mechanisms improves the quality of differential diagnosis and clinical stratification. Future research should focus on integrating early clinical markers for etiological phenotypes of mitral stenosis, prognostic indicators of arrhythmogenic remodeling, and criteria for reversibility of pulmonary hypertension into practical algorithms.

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