

Clinical and Diagnostic Characteristics of Mitral and Aortic Valve Diseases: A Contemporary Review

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How to cite this paper: Hukimovich, T.H., Qizi, Q.N.A. and Qizi, Q.S.A. (2026) Clinical and Diagnostic Characteristics of Mitral and Aortic Valve Diseases: A Contemporary Review. *Journal of Biosciences and Medicines*, 14, 86-95.

<https://doi.org/10.4236/jbm.2026.147008>

Received: May 26, 2026

Accepted: July 4, 2026

Published: July 7, 2026

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Abstract

Valvular heart disease remains a clinically important cause of cardiovascular morbidity, heart failure, arrhythmia, pulmonary hypertension and reduced quality of life. Among cardiac valves, the mitral and aortic valves are the most relevant in acquired left-sided valve disease because they directly determine left ventricular filling, outflow and systemic perfusion. This review compares the anatomical, pathophysiological, diagnostic and therapeutic features of mitral and aortic valve diseases using contemporary guideline-based and peer-reviewed evidence. For transparency, the revised review specifies the databases searched, the search period, core search terms and inclusion/exclusion criteria. It also integrates clinically relevant severity thresholds for mitral stenosis, mitral regurgitation, aortic stenosis and aortic regurgitation. The review shows that mitral stenosis and mitral regurgitation primarily affect left atrial pressure, pulmonary venous circulation and left ventricular volume load, whereas aortic stenosis produces left ventricular outflow obstruction and pressure overload, and aortic regurgitation creates chronic diastolic volume overload. Treatment decisions should not be based only on the name of the affected valve. They must integrate symptoms, lesion severity, ventricular function, valve anatomy, surgical risk, patient age and Heart Valve Team assessment. Contemporary management includes surgical valve repair, surgical valve replacement, transcatheter aortic valve implantation and transcatheter mitral edge-to-edge repair. Accurate differentiation between mitral and aortic valve disease is essential for timely intervention and prevention of irreversible cardiac remodeling.

Keywords

Mitral Valve Disease, Aortic Valve Disease, Valvular Heart Disease,

Echocardiography, Aortic Stenosis, Mitral Regurgitation, TAVI, TEER, Valve Severity Thresholds

1. Introduction

The cardiac valve apparatus is a complex anatomical and functional system that maintains unidirectional blood flow through the heart. The mitral valve is located between the left atrium and the left ventricle. It opens during diastole to allow left ventricular filling and closes during systole to prevent retrograde flow into the left atrium. The aortic valve is located between the left ventricle and the aorta. It opens during systole to allow systemic ejection and closes during diastole to prevent blood from returning into the left ventricle. Because both valves belong to the left side of the heart, their dysfunction has direct consequences for cardiac output, pulmonary circulation and systemic organ perfusion [1] [2].

It is clinically inaccurate to approach mitral and aortic valve diseases as a single uniform entity. Although both affect left-sided cardiac hemodynamics, their mechanisms are substantially different. Mitral stenosis limits blood flow from the left atrium to the left ventricle. Mitral regurgitation causes systolic backflow from the left ventricle to the left atrium. Aortic stenosis forces the left ventricle to generate higher pressure to eject blood through a narrowed valve. Aortic regurgitation allows diastolic backflow from the aorta into the left ventricle and produces chronic volume overload. These mechanisms determine clinical presentation, auscultatory findings, echocardiographic criteria and therapeutic strategy [3] [4].

The topic is relevant for two reasons. First, population ageing has increased the burden of degenerative aortic stenosis and degenerative mitral regurgitation. Second, rheumatic heart disease remains an important cause of mitral valve disease in many low- and middle-income settings [5] [6]. Therefore, a comparative review of mitral and aortic valve disease is useful not only for cardiologists and cardiac surgeons, but also for general physicians, medical educators and researchers who need a structured framework for clinical decision-making. The central question is not simply which valve is affected, but how the lesion changes hemodynamics, when it becomes severe, and which intervention is justified.

2. Methodology

This paper was designed as a narrative clinical review. Literature was searched in PubMed/MEDLINE, Google Scholar, the European Society of Cardiology guideline repository, the American College of Cardiology/American Heart Association guideline resources and major peer-reviewed cardiology journals. The search covered publications from January 2006 to May 2026, with priority given to contemporary guidelines, echocardiographic recommendations, epidemiological studies and major surgical or transcatheter valve trials. Earlier landmark sources were retained only when they remained clinically authoritative or were repeatedly cited

in later guidelines.

The main search terms and Boolean combinations included: “mitral stenosis severity”, “mitral regurgitation EROA regurgitant volume”, “primary secondary mitral regurgitation treatment”, “aortic stenosis valve area mean gradient”, “aortic regurgitation severity echocardiography”, “SAVR versus TAVI guideline”, “transcatheter edge-to-edge repair secondary mitral regurgitation”, “COAPT MITRA-FR” and “mixed valvular heart disease”. Reference lists of relevant guidelines and trials were also screened to identify additional sources directly related to diagnosis, severity grading and intervention selection.

Inclusion criteria were: (1) international or national clinical guidelines on valvular heart disease; (2) echocardiographic recommendation documents defining stenotic or regurgitant lesion severity; (3) peer-reviewed epidemiological studies on acquired valvular heart disease; (4) randomized trials or major clinical studies of SAVR, TAVI, surgical mitral repair/replacement or TEER; and (5) review articles that clarified clinically important differences between mitral and aortic valve lesions. Exclusion criteria were: non-peer-reviewed opinion pieces, conference abstracts without full text, studies unrelated to left-sided valvular disease, articles without direct diagnostic or therapeutic relevance, duplicate publications and sources with incomplete bibliographic information unless they represented essential guideline material.

The analysis was performed in four dimensions: anatomical and functional differences between the mitral and aortic valves; pathophysiological mechanisms of stenosis and regurgitation; diagnostic criteria used in echocardiography and complementary imaging; and principles of surgical and transcatheter treatment selection. No original patient database was used. Therefore, no statistical hypothesis testing, correlation analysis or regression modeling was performed. The article should be interpreted as a narrative clinical review rather than an original observational study.

The main limitation of this methodology is that evidence synthesis was qualitative rather than meta-analytic. The review did not calculate pooled effect sizes or apply formal risk-of-bias scoring. However, it used guideline documents and major clinical trials as the highest-priority evidence sources, which is appropriate for a clinically oriented narrative review intended to compare disease mechanisms and decision points.

3. Literature Review

3.1. Etiological and Epidemiological Context

Contemporary literature classifies valvular heart disease into rheumatic and non-rheumatic etiological groups. Rheumatic disease commonly involves the mitral valve and is strongly associated with mitral stenosis, whereas degenerative calcification is a leading mechanism of aortic stenosis in older adults. Epidemiological studies show that the global burden of valvular disease is shifting: rheumatic heart disease remains concentrated in lower-resource settings, while calcific aortic valve

disease and degenerative mitral valve disease increase with population ageing in higher-income settings [7] [8].

3.2. Mitral Stenosis

In mitral stenosis, the literature emphasizes mitral valve area, mean transmitral gradient, left atrial enlargement, pulmonary hypertension and atrial fibrillation risk. In rheumatic mitral stenosis, leaflet thickening, commissural fusion, subvalvular fibrosis and calcification progressively reduce the mitral orifice. This increases left atrial pressure, causes pulmonary venous congestion and produces exertional dyspnea. In advanced cases, atrial fibrillation, thromboembolism and right-sided heart involvement may develop [9] [10].

Clinically significant severe rheumatic mitral stenosis is generally defined by a mitral valve area of 1.5 cm² or less. A mean transmitral gradient above 5 - 10 mmHg, especially in the correct clinical and heart-rate context, supports hemodynamic significance, but gradient alone is flow-dependent and should not replace valve area, pulmonary pressure and symptom assessment [11]-[13].

3.3. Mitral Regurgitation

Mitral regurgitation is usually divided into primary and secondary forms. In primary mitral regurgitation, the pathology is located in the valve leaflets, chordae tendineae, papillary muscles or annulus. In secondary mitral regurgitation, the valve leaflets may be structurally normal, but left ventricular dilation, dysfunction or regional wall motion abnormalities prevent adequate leaflet coaptation. This distinction is decisive because primary mitral regurgitation is often anatomically suitable for durable surgical repair, whereas secondary mitral regurgitation requires optimized heart failure therapy before transcatheter or surgical intervention is considered [14] [15].

Severe mitral regurgitation is supported by an integrated echocardiographic profile rather than one isolated number. Common severe thresholds include vena contracta width of at least 0.7 cm, effective regurgitant orifice area of at least 0.40 cm² in primary mitral regurgitation, regurgitant volume of at least 60 mL and regurgitant fraction of at least 50%. In secondary mitral regurgitation, quantitative thresholds must be interpreted with left ventricular size, left ventricular ejection fraction, pulmonary pressure and guideline-directed medical therapy status because regurgitation severity and ventricular disease are tightly interdependent [16].

3.4. Aortic Stenosis

Aortic stenosis is characterized in the literature by a long asymptomatic period, progressive left ventricular hypertrophy and worsening prognosis after symptom onset. The classical symptoms of severe aortic stenosis include exertional dyspnea, angina and syncope. Severe high-gradient aortic stenosis is defined by a peak aortic jet velocity of at least 4.0 m/s, mean transvalvular gradient of at least 40 mmHg

and aortic valve area of 1.0 cm² or less, or indexed valve area of 0.6 cm²/m² or less. In low-flow low-gradient states, severity must be confirmed by flow status, dobutamine stress echocardiography when appropriate, valve calcification and clinical context [17].

3.5. Aortic Regurgitation

Aortic regurgitation differs because chronic left ventricular volume overload can be compensated for many years through ventricular dilation. Once symptoms, left ventricular dysfunction or excessive ventricular enlargement appear, delayed intervention increases the risk of irreversible myocardial damage. Severe aortic regurgitation is suggested by an integrated profile that may include vena contracta width greater than 0.6 cm, effective regurgitant orifice area of at least 0.30 cm², regurgitant volume of at least 60 mL, regurgitant fraction of at least 50%, holodiastolic flow reversal in the descending aorta and progressive left ventricular dilation [18].

3.6. Mixed and Combined Valve Disease

Mixed and combined valve disease creates a diagnostic problem because one lesion can alter the apparent severity of another. For example, severe mitral regurgitation may reduce forward stroke volume across the aortic valve and make aortic stenosis appear low-gradient despite anatomically severe obstruction. Conversely, aortic stenosis can increase left ventricular systolic pressure and intensify the regurgitant driving force across an incompetent mitral valve. In combined stenotic and regurgitant disease, valve area, gradients, regurgitant volume and ventricular remodeling must therefore be interpreted together rather than as isolated measurements. This is why multimodality imaging and Heart Valve Team assessment are especially important in discordant or multi-valve disease. Table 1 shows the comparative anatomical and functional characteristics of the mitral and aortic valves.

Table 1. Comparative characteristics of the mitral and aortic valves.

Criterion	Mitral valve	Aortic valve
Anatomical position	Between the left atrium and left ventricle	Between the left ventricle and aorta
Main function	Allows diastolic filling and prevents systolic backflow	Allows systolic ejection and prevents diastolic backflow
Common pathology	Mitral stenosis, mitral regurgitation	Aortic stenosis, aortic regurgitation
Dominant hemodynamic effect	Left atrial pressure/volume load and pulmonary congestion	Left ventricular pressure or volume load and impaired systemic perfusion
Typical risk	Atrial fibrillation, pulmonary hypertension, thromboembolism	Syncope, angina, left ventricular hypertrophy, systolic dysfunction

Table 2 presents the clinically relevant severity thresholds for major left-sided valve lesions.

Table 2. Clinically relevant severity thresholds for major left-sided valve lesions.

Valve lesion	Major severe threshold(s)	Supportive findings	Clinical caution
Mitral stenosis	Mitral valve area $\leq 1.5 \text{ cm}^2$	Mean gradient usually $>5 - 10 \text{ mmHg}$; enlarged LA; pulmonary hypertension	Gradient is heart-rate and flow dependent.
Mitral regurgitation	VC $\geq 0.7 \text{ cm}$; EROA $\geq 0.40 \text{ cm}^2$ in primary MR; RVol $\geq 60 \text{ mL}$; RF $\geq 50\%$	LA/LV dilation; pulmonary venous systolic flow reversal	Secondary MR must be interpreted with LV size, LVEF and GDMT status.
Aortic stenosis	Vmax $\geq 4.0 \text{ m/s}$; mean gradient $\geq 40 \text{ mmHg}$; AVA $\leq 1.0 \text{ cm}^2$ or indexed AVA $\leq 0.6 \text{ cm}^2/\text{m}^2$	Valve calcification; reduced AVA; symptoms; low-flow assessment	Low-flow low-gradient AS requires integrated confirmation.
Aortic regurgitation	VC $> 0.6 \text{ cm}$; EROA $\geq 0.30 \text{ cm}^2$; RVol $\geq 60 \text{ mL}$; RF $\geq 50\%$	Holodiastolic flow reversal; LV dilation; aortic root disease	LV size and symptoms determine timing of intervention.

3.7. Synthesis of Evidence

The comparative analysis indicates that mitral and aortic valve diseases may share symptoms but differ in their hemodynamic origin. Dyspnea, fatigue, palpitations, chest discomfort and reduced exercise tolerance may occur in both groups. In mitral disease, symptoms are often driven by elevated left atrial pressure, pulmonary venous congestion and atrial rhythm disturbances. In aortic disease, symptoms are more closely related to left ventricular outflow obstruction, pressure overload, volume overload and impaired systemic perfusion.

Table 3. Diagnostic and therapeutic approaches in major valve diseases.

Disease	Key echocardiographic criteria	Therapeutic approach
Mitral stenosis	Mitral valve area, mean transmitral gradient, left atrial size, pulmonary artery pressure and valve morphology	Percutaneous mitral commissurotomy when anatomy is favorable and MR is not significant; surgery when anatomy is unfavorable, calcification is severe or another cardiac operation is required.
Mitral regurgitation	EROA, regurgitant volume, vena contracta, left atrial/ventricular size, pulmonary venous flow and pulmonary pressure	Primary MR: repair is preferred when durable repair is likely. Secondary MR: optimize GDMT/CRT first, then consider TEER or surgery in selected patients.
Aortic stenosis	Aortic valve area, peak velocity, mean gradient, LVEF, stroke volume index and valve calcification	SAVR or TAVI based on age, life expectancy, anatomy, procedural risk, vascular access, valve durability and Heart Valve Team decision.
Aortic regurgitation	Regurgitant fraction, vena contracta, pressure half-time, descending aortic flow reversal, aortic root and LV dimensions	Surgery/replacement when symptoms, LV systolic dysfunction, marked LV enlargement or associated aortic root indication appears.

A key diagnostic conclusion is that decision-making should not rely on a single parameter. In aortic stenosis, valve area alone or gradient alone may be misleading; flow status, left ventricular ejection fraction, valve calcification and symptoms

must be integrated. In mitral regurgitation, one isolated parameter such as vena contracta or regurgitant volume is insufficient; left atrial size, left ventricular remodeling, pulmonary venous flow and clinical status must also be assessed.

Therapeutically, the main difference is that mitral valve repair is preferred when anatomy is suitable, whereas severe aortic stenosis is most often treated by valve replacement. Transcatheter aortic valve implantation has expanded from high-risk patients to selected intermediate- and low-risk populations depending on age, anatomy and guideline-based decision-making [18] [19]. In mitral regurgitation, transcatheter edge-to-edge repair has become particularly relevant for selected patients with high surgical risk or secondary mitral regurgitation who remain symptomatic despite optimized heart failure therapy and who meet anatomical and clinical criteria [20] [21]. **Table 3** summarizes the main diagnostic and therapeutic approaches used in major mitral and aortic valve diseases.

4. Discussion

A comparative approach to mitral and aortic valve disease answers three practical questions: which valve is affected, whether the lesion is stenotic or regurgitant, and whether compensatory mechanisms have failed. Clinical errors often occur because symptoms overlap. Dyspnea may be present in mitral stenosis, mitral regurgitation, aortic stenosis and aortic regurgitation, but the mechanism and treatment are different. Auscultation, electrocardiography and chest radiography remain useful, but echocardiography is the central tool for defining lesion type and severity.

The management of primary and secondary mitral regurgitation must be separated clearly. In severe primary mitral regurgitation, the main decision is whether a durable repair can be achieved safely; repair in an experienced valve center is preferred when anatomy and surgical risk are favorable. In severe secondary mitral regurgitation, the first decision is not immediate valve correction but optimization of guideline-directed heart failure therapy, including device therapy when indicated. TEER is most appropriate when severe symptoms persist, anatomy is suitable, surgical risk is high or prohibitive, and the regurgitation appears clinically important relative to the degree of left ventricular remodeling [20] [21].

The choice between SAVR and TAVI in aortic stenosis also requires explicit decision points. SAVR is generally favored in younger patients with longer life expectancy, low surgical risk, bicuspid anatomy requiring repair, associated coronary or aortic surgery needs, or anatomy unsuitable for transfemoral TAVI. TAVI is favored in older patients, patients with high or prohibitive surgical risk, frailty, suitable transfemoral access and anatomy compatible with safe transcatheter implantation. In borderline cases, the Heart Valve Team should integrate age, comorbidity, life expectancy, valve durability, coronary access after implantation and patient preference.

Different results in the TEER trials for secondary mitral regurgitation can be explained mainly by patient selection and disease profile. COAPT enrolled pa-

tients with symptomatic heart failure and significant secondary mitral regurgitation despite maximally tolerated guideline-directed therapy, with generally smaller ventricular volumes and a greater regurgitant burden relative to left ventricular size. MITRA-FR included a broader population with larger ventricles and less disproportionate regurgitation, so the valve lesion may have been more a marker of advanced ventricular disease than an independent therapeutic target. Therefore, the apparent conflict between the trials does not mean TEER is ineffective; it means that benefit depends on selecting patients in whom mitral regurgitation is an actionable driver of decompensation rather than only a consequence of end-stage ventricular remodeling [21].

The strength of this review is its clinically oriented comparison of left-sided valve diseases, including thresholds and intervention logic. Its weakness is that it remains a narrative review without original local patient data. The smallest realistic future improvement would be to collect anonymized echocardiographic data from one hospital or clinic, classify patients into mitral and aortic disease groups, and compare age, sex, lesion type, severity, symptoms, left ventricular ejection fraction, pulmonary pressure and treatment decision. This would transform the article from a descriptive review into an original clinical study with clearer novelty.

Treatment must be individualized. A diagnostic label does not automatically dictate one procedure for all patients. A young low-risk patient with severe aortic stenosis may be better served by surgical valve replacement, whereas an elderly high-risk patient may benefit from transcatheter aortic valve implantation. Primary mitral regurgitation may have excellent results with repair in experienced centers, whereas secondary mitral regurgitation requires optimized heart failure therapy before intervention is considered. Therefore, the practical conclusion is that mechanism, severity and patient risk profile matter more than the valve name alone.

5. Conclusion

Mitral and aortic valve diseases are clinically important forms of left-sided valvular heart disease. They may lead to heart failure, pulmonary hypertension, arrhythmia and impaired quality of life if diagnosed or treated late. Mitral valve disease mainly affects left atrial pressure, pulmonary circulation and left ventricular volume load, whereas aortic valve disease affects left ventricular outflow, pressure load, volume load and systemic perfusion. Echocardiography remains the foundation of diagnosis, but multimodality imaging is necessary when findings are discordant or intervention is planned. Treatment decisions must integrate symptoms, ventricular function, valve anatomy, patient age, surgical risk and Heart Valve Team assessment. This revised review strengthens the original comparison by adding reproducible search methodology, clinically useful severity thresholds, discussion of mixed valve disease, clearer treatment pathways for primary and secondary mitral regurgitation, explicit SAVR/TAVI decision points and critical interpretation of TEER evidence in secondary mitral regurgitation.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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