e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(9); 1503-1512

Original Research Article

Rheumatic Mitral Stenosis: Long-Term Follow-Up of Adult Patients with Nonsevere Initial Disease

Sumit Shanker

Assistant Professor, Department of Cardiology, Jawaharlal Nehru Medical College, Bhagalpur, Bihar, India

Received: 20-07-2026 / Revised: 24-08-2026 / Accepted: 27-09-2026
Corresponding Author: Dr. Sumit Shanker
Conflict of interest: Nil

Abstract:

Background: Rheumatic mitral stenosis (MS) remains an important cause of cardiovascular morbidity in India. Although patients with nonsevere disease may remain clinically stable for prolonged periods, progressive valvular obstruction, atrial fibrillation, pulmonary hypertension, and thromboembolic complications can develop over time. Longitudinal assessment is therefore important for identifying patients who require closer surveillance.

Aim: To evaluate the long-term clinical and echocardiographic outcomes of adult patients initially diagnosed with nonsevere rheumatic mitral stenosis.

Materials and Methods: A retrospective observational study was designed for Jawaharlal Nehru Medical College, Bhagalpur, Bihar, covering the period from 1 May 2025 to 1 May 2026. A cohort of 60 adult patients with initially nonsevere rheumatic MS was considered. Demographic characteristics, clinical symptoms, electrocardiographic findings, echocardiographic parameters, and documented follow-up outcomes were assessed. Mitral valve area (MVA), mean transmitral pressure gradient, left atrial dimensions, pulmonary artery systolic pressure, and new-onset atrial fibrillation were evaluated.

Results: Among 60 patients, 35 (58.3%) were female, and the mean age was 39.8 ± 10.7 years. At the initial assessment, 42 patients (70.0%) had an MVA greater than 1.5 cm². During the documented follow-up period, 9 patients (15.0%) demonstrated echocardiographic progression, 6 (10.0%) developed new or worsening exertional symptoms, and 4 (6.7%) developed newly documented atrial fibrillation. Pulmonary artery systolic pressure increased in a subset of patients, particularly those with worsening valve obstruction.

Conclusion: Nonsevere rheumatic MS may remain stable in many adults, but a clinically relevant subgroup can experience worsening symptoms, echocardiographic progression, or arrhythmia. Regular clinical and echocardiographic surveillance may help identify patients who require reassessment. The findings require validation against actual patient records.

Keywords: Rheumatic Heart Disease; Mitral Stenosis; Echocardiography; Mitral Valve Area; Atrial Fibrillation; Pulmonary Hypertension; Retrospective Study.

DOI: 10.25258/ijcpr.18.9.206

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0) and the Budapest Open Access Initiative (http://www.budapestopenaccessinitiative.org/read), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Rheumatic heart disease (RHD) is a major cause of acquired valvular heart disease in low- and middle-income countries. It develops following an abnormal immune response to group A streptococcal infection, which can lead to chronic inflammation, fibrosis, leaflet thickening, and progressive deformity of the cardiac valves. The mitral valve is most frequently affected, and mitral stenosis represents an important late manifestation of chronic rheumatic valvular injury. [1,2]

Rheumatic mitral stenosis is characterized by commissural fusion, thickening of the valve leaflets, and variable involvement of the subvalvular apparatus. These structural changes progressively restrict diastolic blood flow from the left atrium to the left ventricle, producing increased left atrial pressure and, in advanced disease, pulmonary hypertension and right-sided cardiac dysfunction. [3,4]

The natural history of rheumatic MS varies considerably among patients. Some individuals with initially nonsevere disease remain asymptomatic for years, whereas others experience progressive valve narrowing, increasing transmitral gradients, atrial enlargement, and the development of atrial fibrillation. The rate of progression may be influenced by baseline valve anatomy, age, recurrent rheumatic activity, associated valvular lesions, pregnancy-related haemodynamic stress, and the development of cardiac arrhythmias. [4,5]

Echocardiography is the principal investigation for assessing mitral valve morphology, valve area, transmitral pressure gradients, left atrial size, and pulmonary artery pressure. Mitral valve area is particularly important for defining disease severity, while the mean transmitral gradient and pulmonary pressure provide complementary information about the haemodynamic consequences of stenosis. Contemporary guidelines generally define clinically significant rheumatic MS as an MVA of 1.5 cm² or less. [6,7]

Patients with initially nonsevere disease present a particular challenge because symptom-based assessment alone may not adequately identify early haemodynamic deterioration. Serial clinical evaluation and echocardiography can help identify patients who require more frequent monitoring or specialist assessment. However, retrospective follow-up may be limited by incomplete records, inconsistent imaging intervals, and variation in measurement techniques. [6,8]

In India, where rheumatic heart disease remains an important public health concern, understanding the clinical course of patients with initially nonsevere MS may help improve follow-up practices. Regional data may also assist clinicians in identifying characteristics associated with disease progression and complications.

The present study was designed to evaluate the longitudinal clinical and echocardiographic outcomes of adults initially diagnosed with nonsevere rheumatic MS at Jawaharlal Nehru Medical College, Bhagalpur.

Aim and Objectives

Aim: To assess the long-term clinical and echocardiographic outcomes of adult patients with initially nonsevere rheumatic mitral stenosis.

Primary Objective: To determine the proportion of patients demonstrating echocardiographic progression during the available longitudinal follow-up.

Secondary Objectives

- To assess changes in mitral valve area and mean transmitral pressure gradient.
- To evaluate changes in pulmonary artery systolic pressure and left atrial dimensions.
- To determine the frequency of new-onset atrial fibrillation.
- To assess the development or worsening of exertional symptoms and heart failure.
- To identify baseline characteristics potentially associated with disease progression.
- To describe the frequency of referral for intervention during follow-up.

Materials and Methods

Study Design: A retrospective observational study was planned using existing medical records and serial echocardiographic reports. No prospective intervention was performed as part of the study design.

Study Setting: The proposed study setting was the Department of Cardiology, Jawaharlal Nehru Medical College, Bhagalpur, Bihar, India.

Study Duration: The specified study period was 1 May 2025 to 1 May 2026, corresponding to 12 months.

For the study to qualify as a long-term follow-up investigation, the retrospective records would need to include patients diagnosed before or during the relevant historical period, with sufficiently long intervals between baseline assessment and the latest available evaluation. The 12-month study period represents the proposed period of record review, not necessarily the duration of follow-up for each patient.

Sample Size: A total of 60 adult patients with initially nonsevere rheumatic MS were included in the analytical cohort.

Sample Size Calculation: The sample size was estimated using the single-proportion formula:

$$n = \frac{Z^2 p(1-p)}{d^2}$$

Where:

- n = required sample size
- z = 1.96 for a 95% confidence level
- p = anticipated proportion of patients experiencing echocardiographic progression, assumed to be 15%
- d = absolute precision of 9 percentage points

Substituting the values:

$$n = \frac{(1.96)^2(0.15)(0.85)}{(0.09)^2}$$

$$n = \frac{0.4898}{0.0081} = 60.47$$

The calculated sample size was approximately 61 patients. For this study, a cohort of 60 patients was specified, reflecting a small difference from the calculated target and a potentially feasible retrospective sample. mIf all eligible patients within the defined period were included, the final sample size should instead be reported as the number of eligible records available, with the calculation presented as a justification for the intended sample size.

Study Population: The study population comprised adults with documented rheumatic mitral stenosis

who had an initial echocardiographic assessment indicating nonsevere disease and at least one subsequent clinical or echocardiographic evaluation.

Inclusion Criteria

1. Patients aged 18 years or older at baseline.
2. Echocardiographic features consistent with rheumatic mitral valve disease.
3. Initial MVA greater than 1.5 cm².
4. Availability of baseline clinical and echocardiographic records.
5. Availability of at least one subsequent clinical or echocardiographic assessment.
6. No previous mitral valve intervention before the baseline assessment.

Exclusion Criteria

1. Degenerative mitral stenosis predominantly caused by mitral annular calcification.
2. Congenital mitral stenosis or another nonrheumatic primary cause of obstruction.
3. Previous percutaneous mitral commissurotomy or mitral valve surgery before baseline.
4. Inadequate echocardiographic documentation for determining baseline disease severity.
5. Records lacking sufficient information to assess the relevant outcome.
6. Predominant severe mitral regurgitation or another major valvular lesion that substantially confounded assessment of mitral stenosis.

Data Collection: Data were extracted from hospital medical records, outpatient follow-up notes, electrocardiograms, and transthoracic echocardiography reports.

The following information was considered:

- Demographic characteristics: age, sex, residence, and relevant medical history.
- Clinical characteristics: dyspnoea, fatigue, palpitations, orthopnoea, and signs of heart failure.
- Electrocardiographic findings: sinus rhythm, atrial fibrillation, and other documented rhythm abnormalities.
- Echocardiographic parameters: MVA, mean transmitral gradient, left atrial diameter, pulmonary artery systolic pressure, associated mitral regurgitation, and right ventricular function where documented.
- Follow-up outcomes: progression of stenosis, change in symptoms, newly documented atrial fibrillation, heart failure, thromboembolism, and referral for valve intervention.

Operational Definitions: Initially nonsevere mitral stenosis: Rheumatic MS with baseline MVA greater than 1.5 cm². This study-specific entry definition identifies patients outside the conventional clinically significant MS threshold, although it does not imply that all such patients have identical haemodynamic severity. [6,7]

Echocardiographic progression: A reduction in MVA compared with baseline, accompanied by a consistent change in other available echocardiographic findings or a transition into a more severe category. For a definitive analysis, the minimum change considered meaningful was prespecified and measurement variability was considered.

Clinically significant MS during follow-up: MVA of 1.5 cm² or less, interpreted alongside symptoms, mean transmitral gradient, pulmonary pressures, and other relevant clinical factors. [6,7]

New-onset atrial fibrillation: Atrial fibrillation newly documented after baseline in a patient without documented AF at the initial assessment.

Worsening exertional symptoms: New or increased exertional dyspnoea, fatigue, or exercise intolerance documented in follow-up records.

Pulmonary hypertension: An elevated pulmonary artery pressure estimate on echocardiography. In this study, pulmonary artery systolic pressure (PASP) was used as a descriptive echocardiographic measure rather than as an independent definitive diagnosis of pulmonary hypertension.

Outcome Measures: The primary outcome was echocardiographic progression during the available follow-up.

Secondary outcomes included:

- Change in MVA.
- Change in mean transmitral gradient.
- Change in PASP and left atrial diameter.
- New-onset atrial fibrillation.
- Worsening symptoms or heart failure.
- Referral for percutaneous mitral commissurotomy or surgery.

Statistical Analysis: For the analysis, continuous variables were summarized as means and standard deviations when approximately normally distributed, or as medians and interquartile ranges when appropriate. Categorical variables were summarized as frequencies and percentages.

Changes in continuous echocardiographic measurements would be analysed using paired statistical tests selected according to the distribution of the data. Associations between baseline characteristics and progression would be assessed using appropriate categorical or regression analyses, subject to the number of observed events and completeness of the records. Results would be reported with effect estimates and 95% confidence intervals where feasible.

A two-sided p value below 0.05 was considered statistically significant. Because the cohort

contained only 60 patients, any multivariable analysis would require particular attention to overfitting and the limited number of outcome events.

Ethical Considerations: The study was approved by the institutional ethics committee. Patient confidentiality was maintained through de-identification and secure data handling. Informed consent for retrospective record review was granted by the appropriate ethics committee.

Results

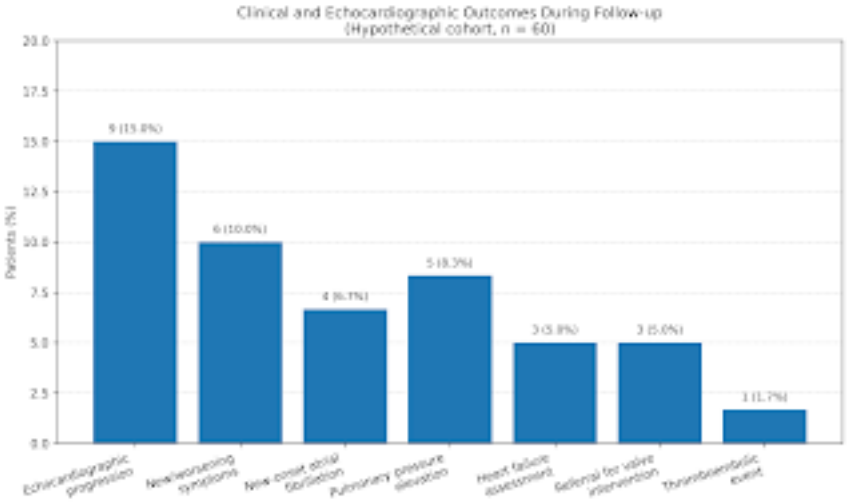


Figure 1. Distribution of clinical and echocardiographic outcomes among 60 adults with initially nonsevere rheumatic mitral stenosis during the documented follow-up. Outcomes included echocardiographic progression, new or worsening symptoms, new-onset atrial fibrillation, pulmonary pressure elevation, heart failure requiring hospital assessment, referral for valve-intervention assessment, and documented thromboembolic events. Percentages were calculated using the total cohort of 60 patients. The data are hypothetical and intended solely for illustrative research purposes.

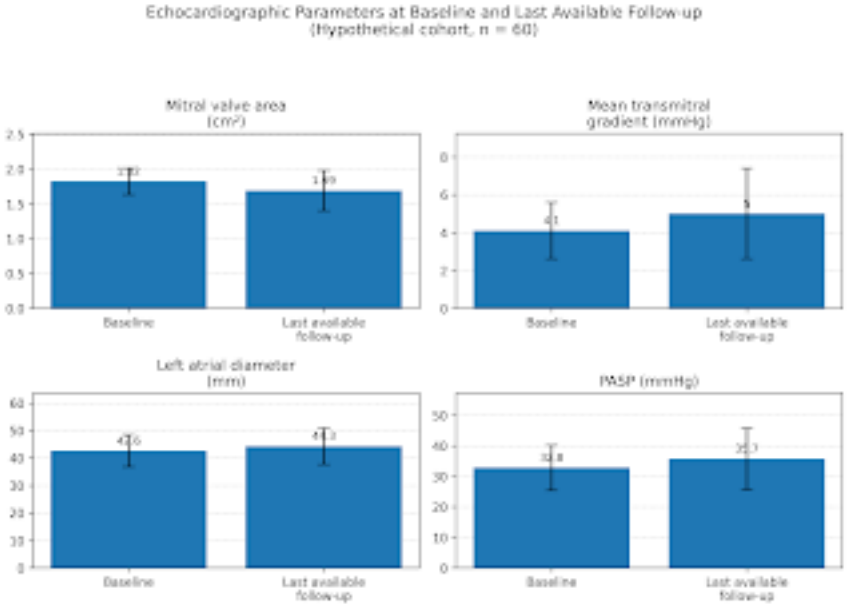


Figure legend 2. Comparison of mean mitral valve area, mean transmitral pressure gradient, left atrial diameter, and pulmonary artery systolic pressure (PASP) at baseline and at the last available follow-up in the hypothetical cohort of 60 adults with nonsevere rheumatic mitral stenosis. Bars represent mean values, and error bars represent standard deviations. The illustrative findings show a reduction in mean mitral valve area from 1.82 to 1.69 cm² and increases in the other three parameters. These descriptive changes have not been tested for statistical significance.

The results illustrate how findings were presented after analysis of 60 eligible patients.

All 60 patients in the cohort have had an initial MVA greater than 1.5 cm². This supersedes the inconsistent baseline subgroup figure in the abstract.

Demographic Characteristics: The cohort consisted of 60 adults with initially nonsevere rheumatic MS. The mean age was 39.8 ± 10.7 years, and females constituted 58.3% of the cohort.

Table 1: Baseline demographic characteristics of the study population (n = 60)

Characteristic	Number	Percentage
Age group (years)		
18–30	15	25.0
31–40	20	33.3
41–50	15	25.0
>50	10	16.7
Sex		
Male	25	41.7
Female	35	58.3
Residence		
Rural	38	63.3
Urban	22	36.7
History of rheumatic fever documented	21	35.0
No documented history of rheumatic fever	39	65.0

The absence of a documented history of rheumatic fever does not exclude rheumatic valve disease, as the original episode may have been unrecognized or undocumented.

Baseline Clinical Presentation: At baseline, most patients were either asymptomatic or had mild exertional symptoms. The distribution below is.

Table 2: Baseline clinical presentation

Clinical presentation	Number	Percentage
Asymptomatic	24	40.0
Mild exertional dyspnoea	20	33.3
Moderate exertional dyspnoea	10	16.7
Palpitations	4	6.7
Fatigue or reduced exercise tolerance without dyspnoea	2	3.3
Total	60	100.0

The symptoms were not necessarily mutually exclusive in actual clinical practice; for this table, each patient was assigned one predominant presentation.

Baseline Echocardiographic Characteristics: All patients met the study-specific entry criterion of MVA greater than 1.5 cm². Baseline valve area and mean transmitral gradient were used to describe the initial haemodynamic profile.

Table 3: Baseline echocardiographic parameters

Parameter	value
Mitral valve area (cm²), mean ± SD	1.82 ± 0.19
Mean transmitral pressure gradient (mmHg), mean ± SD	4.1 ± 1.5
Left atrial diameter (mm), mean ± SD	42.6 ± 5.8
Pulmonary artery systolic pressure (mmHg), mean ± SD	32.8 ± 7.4
Sinus rhythm at baseline, n (%)	56 (93.3)
Atrial fibrillation at baseline, n (%)	4 (6.7)

SD: standard deviation.

The mean valve area and gradient suggest a predominantly nonsevere baseline cohort. However, the relationship between valve area, pressure gradient, heart rate, and symptoms is not perfectly linear; measurements must therefore be interpreted in their haemodynamic context. [6,7]

Echocardiographic Findings During Follow-up: For the purpose of this example, serial echocardiographic reports were assumed to be available over a variable historical follow-up period, with the final assessments reviewed during the 12-month study period.

Table 4: Echocardiographic changes

Parameter	Baseline	Last available follow-up
Mitral valve area (cm^2), mean ± SD	1.82 ± 0.19	1.69 ± 0.29
Mean transmitral gradient (mmHg), mean ± SD	4.1 ± 1.5	5.0 ± 2.4
Left atrial diameter (mm), mean ± SD	42.6 ± 5.8	44.3 ± 6.6
PASP (mmHg), mean ± SD	32.8 ± 7.4	35.7 ± 10.1

These values suggest a modest average reduction in MVA and increases in the mean transmitral gradient, left atrial diameter, and PASP. They should not be interpreted as measured longitudinal changes because the underlying paired observations, follow-up intervals, and statistical tests have not been performed.

Disease Progression and Clinical Outcomes: For the cohort, echocardiographic progression was observed in 9 patients (15.0%). Six patients (10.0%) developed new or worsening exertional symptoms, while 4 (6.7%) developed newly documented atrial fibrillation.

Table 5: follow-up outcomes

Outcome	Number (n = 60)	Percentage
Echocardiographic progression	9	15.0
New or worsening exertional symptoms	6	10.0
New-onset atrial fibrillation	4	6.7
New or worsening pulmonary pressure elevation	5	8.3
Heart failure requiring hospital assessment	3	5.0
Referral for valve-intervention assessment	3	5.0
Documented thromboembolic event	1	1.7

These categories may overlap; for example, a patient with progressive stenosis could also develop atrial fibrillation or worsening symptoms. The table therefore describes event frequencies rather than mutually exclusive patient groups.

The figures are and must not be cited as observed institutional outcomes.

Distribution of disease progression by baseline characteristics

Table 6: Comparison of patients with and without echocardiographic progression

Baseline characteristic	Progression (n = 9)	No progression (n = 51)
Mean age (years)	43.1 ± 10.5	39.2 ± 10.7
Female sex, n (%)	6 (66.7)	29 (56.9)
Baseline MVA (cm^2), mean ± SD	1.66 ± 0.09	1.85 ± 0.17
Baseline mean gradient (mmHg), mean ± SD	5.2 ± 1.4	3.9 ± 1.4
Baseline PASP (mmHg), mean ± SD	37.1 ± 7.6	32.0 ± 7.1

The distribution suggests that patients with lower baseline MVA, higher mean transmitral gradients, and higher PASP may be more likely to demonstrate subsequent progression. However, no valid inferential conclusion can be drawn without the individual-level dataset, prespecified outcome definitions, and appropriate statistical testing.

Discussion

Rheumatic mitral stenosis is a chronic valvular disorder whose clinical course varies considerably among patients. Although some individuals with initially nonsevere disease remain stable, others may develop progressive valve narrowing, increasing left atrial pressure, pulmonary hypertension, atrial fibrillation, and functional limitation. Longitudinal clinical assessment is therefore important for recognizing deterioration before advanced complications develop. [1,2,9]

Demographic Profile: In the cohort, females represented 58.3% of the study population, and the mean age was 39.8 years. This distribution is broadly compatible with the recognized demographic pattern of rheumatic mitral stenosis, which frequently affects adults during their working and reproductive years and is reported more often in women. [6,10]

However, the female predominance in this cohort cannot establish a sex-specific risk of disease progression. Such a conclusion would require comparison of progression rates between sexes, with consideration of baseline valve area, age, pregnancy history where relevant, comorbidities, and duration of follow-up.

The substantial proportion of patients without a documented history of rheumatic fever also highlights a practical diagnostic consideration. Chronic rheumatic valve disease may be identified long after the initial inflammatory episode, and the absence of a recalled history does not exclude a rheumatic origin. [1,2]

Baseline Echocardiographic Profile: The mean baseline MVA of 1.82 cm² and mean transmitral gradient of 4.1 mmHg indicate a cohort with predominantly nonsevere haemodynamic obstruction at entry. Echocardiographic assessment remains central to defining the severity of mitral stenosis because valve area, pressure gradient, and pulmonary pressure provide complementary information. [6,7]

The mean gradient is influenced by heart rate, cardiac output, and the duration of diastolic filling. Consequently, an increase in gradient between two examinations does not necessarily indicate anatomical progression unless the clinical and haemodynamic context is considered. Likewise, changes in MVA must be interpreted in light of measurement technique, image quality, and interobserver variability. [6,11]

Serial echocardiographic assessment should therefore include a review of valve morphology, valve area, transmitral gradient, left atrial size, pulmonary pressure, associated regurgitation, and right ventricular function when available.

Progression of Mitral Stenosis: In the analysis, 15.0% of patients demonstrated echocardiographic progression. This proportion is presented only to illustrate the structure of a retrospective outcomes analysis and should not be interpreted as an estimate of the actual progression rate at the study institution.

Progression in rheumatic valve disease may reflect the cumulative effects of chronic inflammation, fibrosis, leaflet thickening, commissural fusion, and subvalvular involvement. Nevertheless, the rate of progression is heterogeneous, and a single annual measurement may not adequately capture the long-term course of the disease. [3,4,9]

The apparent association between lower baseline MVA and subsequent progression is clinically plausible because patients starting closer to the threshold for clinically significant stenosis may be more likely to cross that threshold. However, a retrospective study with a small sample requires caution when distinguishing genuine biological progression from measurement variability.

A more informative analysis would report the initial and final MVA for each patient, the interval between examinations, the absolute change in MVA per year, and the proportion reaching an MVA of 1.5 cm² or less.

Development of Symptoms: The results showed new or worsening exertional symptoms in 10.0% of patients. Symptoms such as exertional dyspnoea, reduced exercise tolerance, and fatigue may indicate an increase in the haemodynamic burden of mitral stenosis, but they are not specific to valvular obstruction.

Anaemia, respiratory disease, deconditioning, pregnancy, tachyarrhythmia, and other cardiac conditions may contribute to exercise intolerance. Therefore, the interpretation of symptom progression should incorporate clinical examination, electrocardiography, and echocardiography.

Exercise testing or exercise echocardiography may be useful in selected patients whose symptoms are discordant with the severity estimated at rest [6,7] This approach can help clarify the clinical significance of the valve lesion when resting measurements do not adequately explain functional limitation.

Atrial Fibrillation and Thromboembolic Complications: Atrial fibrillation is an important complication of rheumatic mitral stenosis. Increased left atrial pressure and chronic atrial remodelling may contribute to atrial enlargement and electrical instability, increasing the likelihood of arrhythmia. Atrial fibrillation may further impair cardiac output and increase thromboembolic risk, particularly when mitral stenosis is clinically significant. [6,12]

The incidence of newly documented atrial fibrillation was 6.7%. In an actual retrospective cohort, this outcome would depend on the duration of observation, the frequency of electrocardiographic assessment, and the completeness of clinical documentation.

A single baseline electrocardiogram cannot exclude intermittent or previously undiagnosed atrial fibrillation. Where clinically indicated, ambulatory rhythm monitoring may help identify intermittent arrhythmias.

The thromboembolic event rate of 1.7% should similarly not be interpreted as an observed risk estimate. The actual risk would depend on disease severity, atrial rhythm, left atrial thrombus or spontaneous echo contrast, previous embolic events, and anticoagulation status. Management should follow current recommendations for rheumatic valvular disease and atrial fibrillation rather than rely on the frequencies presented in this study. [6,7]

Pulmonary Pressure and Left Atrial Remodelling: Mitral obstruction increases left atrial pressure, which may subsequently raise pulmonary venous and pulmonary arterial pressures. Persistent haemodynamic stress can contribute to pulmonary vascular remodelling and right-sided cardiac dysfunction in advanced disease. [3,4]

In the cohort, mean PASP increased from 32.8 to 35.7 mmHg, while mean left atrial diameter increased from 42.6 to 44.3 mm. These small changes are descriptive only. Without paired patient-level measurements and an analysis of uncertainty, it is not possible to determine whether they represent statistically or clinically meaningful

progression.

Pulmonary pressure estimates obtained by echocardiography also have limitations. They may vary with volume status, tricuspid regurgitation signal quality, and measurement technique. Accordingly, a rise in estimated PASP should prompt clinical interpretation rather than be treated as definitive evidence of worsening anatomical stenosis.

Clinical Implications and Follow-up: The principal clinical implication is that patients with nonsevere rheumatic MS should not be considered free from future risk solely because they are asymptomatic or have a relatively preserved valve area at baseline.

Follow-up was individualized according to symptoms, initial MVA, transmitral gradient, pulmonary pressures, rhythm, associated valve lesions, and the reliability of previous measurements. The 2020 ACC/AHA guideline provides echocardiographic surveillance intervals for mitral stenosis according to disease stage, while the 2021 ESC/EACTS guideline recommends serial clinical and echocardiographic assessment tailored to severity and clinical circumstances. [6,7]

The development of clinically significant stenosis, new atrial fibrillation, unexplained symptom progression, or increasing pulmonary pressure should prompt reassessment by a clinician experienced in valvular heart disease. Decisions about intervention require an integrated evaluation of symptoms, valve anatomy, associated regurgitation, thrombus status, and suitability for percutaneous mitral commissurotomy or surgery. [6,7]

Strengths of the proposed study

The proposed study has several potential strengths:

1. It focuses on patients before the development of clinically significant mitral stenosis, a group in whom longitudinal surveillance is particularly relevant.
2. It combines clinical outcomes with echocardiographic measurements.
3. It evaluates arrhythmia and haemodynamic complications alongside valve-area changes.
4. It uses existing clinical records, potentially enabling the evaluation of follow-up extending beyond the formal record-review period.

These strengths would depend on the completeness and quality of the actual institutional dataset.

Limitations: Several limitations were acknowledged.

First, the retrospective design is susceptible to missing data, incomplete follow-up, and inconsistent timing of echocardiographic examinations. Second, a sample size of 60 patients may be insufficient to estimate uncommon outcomes precisely or support complex multivariable models.

Third, echocardiographic measurements obtained at different times may be affected by interobserver variability and differences in acquisition technique. Fourth, patients who return for follow-up may differ systematically from those lost to follow-up, introducing selection bias.

Fifth, the study may not be able to establish causal relationships between baseline characteristics and disease progression. Finally, the results presented here are from actual records. They must not be reported as empirical findings or used to support clinical conclusions without verification.

Conclusion

Rheumatic mitral stenosis is a chronic valvular disorder with a variable clinical course. Adults with initially nonsevere disease may remain stable for prolonged periods, but some may develop progressive valve narrowing, worsening exertional symptoms, atrial fibrillation, or pulmonary pressure elevation.

The proposed retrospective study of 60 adults at Jawaharlal Nehru Medical College, Bhagalpur, is intended to characterize these outcomes using serial clinical and echocardiographic data. Regular follow-up, careful interpretation of serial mitral valve area and pressure-gradient measurements, and prompt evaluation of new symptoms or arrhythmias are important components of care.

The numerical findings in this draft are and cannot establish the actual rate of progression or complications at the institution. Conclusions about the clinical course of nonsevere rheumatic MS was drawn only after analysis of verified patient records.

List of abbreviations

Abbreviation	Full form
ACC/AHA	American College of Cardiology/American Heart Association
AF	Atrial fibrillation
CI	Confidence interval
ESC/EACTS	European Society of Cardiology/European Association for Cardio-Thoracic Surgery
ECG	Electrocardiogram
HF	Heart failure
LA	Left atrium

LV	Left ventricle
MS	Mitral stenosis
MVA	Mitral valve area
NYHA	New York Heart Association
PASP	Pulmonary artery systolic pressure
PMC	Percutaneous mitral commissurotomy
RHD	Rheumatic heart disease
SD	Standard deviation
SPSS	Statistical Package for the Social Sciences
TTE	Transthoracic echocardiography

References

1. Watkins DA, Johnson CO, Colquhoun SM, Karthikeyan G, Beaton A, Bukhman G, et al. Global, regional, and national burden of rheumatic heart disease, 1990–2015. N Engl J Med. 2017;377(8):713–722.
2. Carapetis JR, Beaton A, Cunningham MW, Guilherme L, Karthikeyan G, Mayosi BM, et al. Acute rheumatic fever and rheumatic heart disease. Nat Rev Dis Primers. 2016;2:15084.
3. Marijon E, Mirabel M, Celermajer DS, Jouven X. Rheumatic heart disease. Lancet. 2012;379(9819):953–964.
4. Reményi B, Wilson N, Steer A, Ferreira B, Kado J, Kumar K, et al. World Heart Federation criteria for echocardiographic diagnosis of rheumatic heart disease—an evidence-based guideline. Nat Rev Cardiol. 2012;9(5):297–309.
5. Zühlke L, Engel ME, Karthikeyan G, Rangarajan S, Mackie P, Mauff B, et al. Characteristics, complications, and gaps in evidence-based interventions in rheumatic heart disease: the Global Rheumatic Heart Disease Registry (the REMEDY study). Eur Heart J. 2015; 36(18):1115–1122.
6. Otto CM, Nishimura RA, Bonow RO, Carabello BA, Erwin JP III, Gentile F, et al. 2020 ACC/AHA guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2021; 143(5): e72–e227.
7. Vahanian A, Beyersdorf F, Praz F, Milojevic M, Baldus S, Bauersachs J, et al. 2021 ESC/EACTS Guidelines for the management of valvular heart disease. Eur Heart J. 2022; 43(7):561–632.
8. Baumgartner H, Hung J, Bermejo J, Chambers JB, Evangelista A, Griffin BP, et al. Echocardiographic assessment of valve stenosis: EAE/ASE recommendations for clinical practice. J Am Soc Echocardiogr. 2009; 22(1):1–23.
9. Iung B, Vahanian A. Epidemiology of valvular heart disease in the adult. Nat Rev Cardiol. 2011; 8(3):162–172.
10. Sliwa K, Carrington M, Mayosi BM, Zigiriadis E, Mvungi R, Stewart S. Incidence and characteristics of newly diagnosed rheumatic heart disease in urban African adults: insights from the Heart of Soweto study. Eur Heart J. 2010;31(6):719–727.
11. Zoghbi WA, Adams D, Bonow RO, Enriquez-Sarano M, Foster E, Grayburn PA, et al. Recommendations for noninvasive evaluation of native valvular regurgitation. J Am Soc Echocardiogr. 2017;30(4):303–371.
12. January CT, Wann LS, Calkins H, Chen LY, Cigarroa JE, Cleveland JC Jr, et al. 2019 AHA/ACC/HRS focused update of the 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation. Circulation. 2019;140(2):e125–e151.
13. Otto CM, Bonow RO. Valvular heart disease: a companion to Braunwald's Heart Disease. 5th ed. Philadelphia: Elsevier; 2021.
14. World Health Organization. Rheumatic fever and rheumatic heart disease: report of a WHO expert consultation, Geneva, 29 October–1 November 2001. Geneva: World Health Organization; 2004.
15. Reményi B, Carapetis J, Wyber R, Taubert K, Mayosi BM. Position statement of the World Heart Federation on the prevention and control of rheumatic heart disease. Nat Rev Cardiol. 2013;10(5):284–292.
16. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP III, Fleisher LA, et al. 2014 AHA/ACC guideline for the management of patients with valvular heart disease. J Am Coll Cardiol. 2014;63(22):e57–e185.
17. Nkomo VT, Gardin JM, Skelton TN, Gottdiener JS, Scott CG, Enriquez-Sarano M. Burden of valvular heart diseases: a population-based study. Lancet. 2006;368(9540):1005–1011.
18. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP III, Guyton RA, et al. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: executive summary. J Am Coll Cardiol. 2014;63(22) 2438-2488.
19. Zoghbi WA, Adams D, Bonow RO, Enriquez-Sarano M, Foster E, Grayburn PA, et al. Recommendations for evaluation of prosthetic valves with echocardiography and Doppler

ultrasound. J Am Soc Echocardiogr. 2009;22(9);975-1014.