IMPACT OF SURGICAL TREATMENT ON INTRACARDIAC HEMODYNAMIC PARAMETERS: A COMPARATIVE ANALYSIS IN PATIENTS WITH CHRONIC HEART FAILURE DEVELOPED AGAINST THE BACKGROUND OF RHEUMATIC MITRAL VALVE DISEASE

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https://doi.org/10.5281/zenodo.18888810

ARTICLE INFO

Received: 01st March 2026
Accepted: 04th March 2026
Online: 06th March 2026

KEYWORDS

Surgical correction of mitral valve disease is the most effective method for eliminating major hemodynamic disturbances

ABSTRACT

According to the World Health Organization, nearly 33 million people worldwide suffer from chronic rheumatic heart disease, and annually over 300,000 deaths are associated with this disease [1]. Among rheumatic heart defects, mitral valve involvement is the most common, which, when prolonged, leads to the development of chronic heart failure (CHF)..

Introduction. According to the World Health Organization, nearly 33 million people worldwide suffer from chronic rheumatic heart disease, and annually over 300,000 deaths are associated with this disease [1]. Among rheumatic heart defects, mitral valve involvement is the most common, which, when prolonged, leads to the development of chronic heart failure (CHF).

Rheumatic mitral valve disease (RMVD) results in increased pressure-volume load on the left atrium, leading to left atrial remodeling — dilation and fibrosis [2,3]. According to Roitberg and Strunsky, mitral valve defect is considered the first hemodynamic barrier, obstructing blood flow between the left atrium and left ventricle [23]. Consequently, pressure in the left atrium increases, subsequently transmitted through the pulmonary veins to the pulmonary artery, leading to the development of a second hemodynamic barrier — pulmonary hypertension. This, in turn, increases the load on the right heart chambers and exacerbates the course of CHF.

Surgical correction of mitral valve disease is the most effective method for eliminating major hemodynamic disturbances. However, even after surgical intervention, some patients exhibit persistent left atrial enlargement and atrial fibrillation. This indicates the presence of irreversible aspects of remodeling processes [4,5].

The aim of this study is to evaluate the impact of surgical treatment on intracardiac hemodynamic parameters and to identify residual pathological changes in the postoperative

period in patients with chronic heart failure developed against the background of rheumatic mitral valve disease.

Materials and methods: The study was conducted at the Namangan branch of the Republican Specialized Scientific-Practical Medical Center of Cardiology during 2022–2025. This prospective, comparative, cross-sectional study involved 90 patients with CHF developed against the background of RMVD. All patients were divided into two groups based on the morphological status of the mitral valve: Group I (n=45) — patients who underwent surgical intervention (valve replacement or commissurotomy) for rheumatic mitral valve disease within the last 5 years. Group II (n=45) — patients with persistent rheumatic mitral valve disease who did not undergo surgical treatment. Patients aged 33 to 79 years (mean age 58±2.97 years) with CHF developed against the background of RMVD were included. Patients with the following criteria were excluded from the study: non-rheumatic etiology of heart valve disease, acute myocardial infarction or cerebrovascular accident within the last 3 months, severe renal failure (GFR <30 ml/min/1.73 m^2), and predicted life expectancy of less than 3 years.

Examination methods. The following examinations were performed in all patients:

Echocardiography (EchoCG) — performed using a Mindray I 90 device with a 2.5 MHz transducer in M-mode, B-mode, and Doppler modes (pulse-wave, continuous-wave, color scanning). Left ventricular ejection fraction (LVEF) was determined using the Simpson method in apical 4- and 2-chamber views. Left atrial size (LA size) was measured in the parasternal long-axis view. Mitral valve diastolic pressure gradient (MV PG) was determined by Doppler echocardiography. Systolic pulmonary artery pressure (SPAP) was calculated based on the maximum velocity of the tricuspid regurgitation jet. Left ventricular myocardial mass (LVMM) was calculated using the Devereux formula. Electrocardiography (ECG) — performed using a Schiller D 40/7 device in 12-channel mode. The presence of permanent atrial fibrillation (AF) was assessed. Assessment of clinical status: Score according to the modified SHOKS scale (scale for assessing the severity of clinical condition in heart failure) by B.Y Mareev; Minnesota Living with Heart Failure Questionnaire (MLHFQ); 6-minute walk test (6-MWT). Statistical analysis. Data were processed using Microsoft Office Excel 2013 and Statistica for Windows v10 (StatSoft, USA) software. Mean values (M) and standard errors (±m) were calculated. Student's t-test (for independent samples) was used to assess differences between groups, and the Z-test was used to compare relative indicators. Differences were considered statistically significant at $p<0.05$. Ethical issues**.** The study was approved by the Ethics Committee of the Republican Specialized Scientific-Practical Medical Center of Cardiology. Informed consent was obtained from all patients.

Results: Of the 90 patients included in the study, 45 (50%) were in Group I and 45 (50%) in Group II. No statistically significant differences in gender and age were found between the groups. In Group I, there were 29 females (64.4%) and 16 males (35.6%), while in Group II, there were 36 females (80.0%) and 9 males (20.0%) (p=0.163). The mean age was 60.2±1.69 years in Group I and 56.1±1.25 years in Group II (p=0.054).

The duration of CHF was significantly longer in Group I (6.7±0.8 years vs. 4.2±0.7 years in Group II; p=0.02), indicating a longer disease history in surgically treated patients. Significant differences were observed in the distribution of heart failure functional class (FC): in Group I, 28 patients (62.2%) were in FC I, while in Group II, FC II (21 patients; 46.7%) and FC III (14 patients; 31.1%) predominated (p<0.001 and p=0.015, respectively). Analysis of Echocardiographic Parameters. Both groups exhibited the phenotype of preserved left ventricular ejection fraction. However, LVEF was statistically significantly higher in Group II (60.8±1.56% vs. 55.8±0.98% in Group I; p=0.008). The main hemodynamic differences were as follows:

Left Atrial Size (LA size) — significantly larger in Group II (56.7±0.81 mm) compared to Group I (45.8±0.64 mm; p<0.001). This indicates ongoing left atrial dilation in patients with persistent mitral valve disease. Mitral Valve Diastolic Pressure Gradient (MV PG) — near-normal in Group I (11.2±0.52 mmHg), while remaining elevated in Group II (16.3±1.16 mmHg; p<0.001). This confirms that surgery effectively eliminated the hemodynamic barrier at the mitral valve level. Systolic Pulmonary Artery Pressure (SPAP) — 29.8±1.2 mmHg in Group I vs. 42.1±1.76 mmHg in Group II (p<0.001). Patients who underwent surgical treatment showed regression of pulmonary hypertension. Right Ventricular Systolic Pressure (RVSP) — higher in Group II (42.5±1.89 mmHg) compared to Group I (35.1±1.50 mmHg; p=0.002), indicating persistent load on the right heart chambers. Tricuspid Valve Regurgitation (TVR) — slightly higher in Group II (1.5±0.08) compared to Group I (1.2±0.09; p=0.01). Other morphometric parameters of the left ventricle (LV EDD, LV EDV, LV ESV, LVMM) did not show statistically significant differences between the groups (p>0.05).

Rhythm Disturbances and Clinical Status: Permanent Atrial Fibrillation (AF) was significantly more common in Group I — observed in 34 patients (75.5%), compared to 15 patients (33.3%) in Group II (p<0.001). This indicates a persistently high risk of arrhythmia even in surgically treated patients.

Clinical parameters: SHOKS score: 1.8±0.14 in Group I vs. 3.6±0.11 in Group II (p<0.001);

Minnesota Questionnaire score: 15.6±0.87 in Group I vs. 32.9±0.98 in Group II (p<0.001);

6-minute walk test: 428.9±9.8 m in Group I vs. 312.1±11.9 m in Group II (p<0.001).

These data confirm that surgically treated patients have a better clinical status and higher quality of life.

Analysis of Biomarkers**.** NT-proBNP levels were 804.91±115.0 pg/ml in Group I and 1240.91±165.0 pg/ml in Group II (p=0.031**).** C-reactive protein (CRP) levels were 12.7±0.87 mg/ml in Group I and 16.7±1.34 mg/ml in Group II (p=0.014). Both biomarkers were statistically significantly higher in Group II, indicating ongoing neurohumoral activation and inflammatory processes.

Discussion. The results of our study demonstrate that surgical treatment in patients with CHF developed against the background of RMVD effectively corrects major hemodynamic disturbances (pulmonary hypertension, high mitral gradient). This is particularly evident in the normalization of pulmonary artery pressure and improvement in patients' functional status. Our observations confirm the concept of hemodynamic barriers by Roitberg and Strunsky [23]. Surgical treatment eliminates the first hemodynamic barrier (at

the mitral valve level), leading to regression of the second barrier (pulmonary hypertension). Furthermore, according to the study by Serge C. Harb and Brian P. Griffin, in mitral stenosis, a pressure gradient appears when the orifice area narrows to 2 cm^2, and tachycardia exacerbates this condition [13]. In our study, Group II also exhibited a higher heart rate (91.7±3.07 bpm), contributing to the increased pressure gradient.

The most significant finding of our study is the persistently high prevalence of atrial fibrillation (75.5%) in the surgically treated group, despite improved hemodynamic parameters. This phenomenon is explained by the "remodeling begets arrhythmia" concept proposed by A.V. Bogachev-Prophiev and colleagues [14]. Prolonged left atrial dilation leads to structural (fibrosis) and electrophysiological remodeling, and these changes do not fully reverse even after the hemodynamic barrier is eliminated. As noted by January et al. [14], atrial fibrillation is a natural stage of mitral valve disease, occurring in 30–84% of patients, and our results fall within this range.

The study by Estu Rudiktiyo et al. [16] showed that RMVD predominantly affects females (84%) and occurs with preserved left ventricular ejection fraction. In our study, females constituted 71.1%, and left ventricular ejection fraction was preserved (p=0.008) — consistent with the heart failure with preserved ejection fraction (HFpEF) phenotype. The elevated NT-proBNP levels in both groups (804.91 pg/ml in Group I and 1240.91 pg/ml in Group II) are noteworthy. These values are higher than the average level (986 pg/ml) reported in the REDHOT study [22] for patients hospitalized with dyspnea, indicating higher neurohumoral activation in rheumatic etiology CHF.

Conclusion

Surgical treatment in patients with chronic heart failure developed against the background of rheumatic mitral valve disease effectively corrects major hemodynamic disturbances: it significantly reduces systolic pulmonary artery pressure (from 42.1±1.76 mmHg to 29.8±1.2 mmHg) and the mitral diastolic gradient (from 16.3±1.16 mmHg to 11.2±0.52 mmHg).

As a result of hemodynamic improvement, surgically treated patients show significant improvement in clinical status (SHOKS score from 3.6±0.11 to 1.8±0.14), exercise tolerance (6-MWT from 312.1±11.9 m to 428.9±9.8 m), and quality of life (Minnesota score from 32.9±0.98 to 15.6±0.87).

However, surgical treatment does not completely reverse pathological changes formed in the left atrium: although left atrial size in Group I (45.8±0.64 mm) is smaller compared to Group II, it does not return to normal levels.

Atrial fibrillation is more common in surgically treated patients (75.5% vs. 33.3% in Group II; $p<0.001$), indicating the irreversible nature of structural (fibrosis) and electrical remodeling resulting from prolonged left atrial dilation.

The elevated levels of NT-proBNP and CRP even in the surgically treated group (804.91±115.0 pg/ml and 12.7±0.87 mg/ml, respectively) indicate incomplete suppression of neurohumoral activity and inflammatory processes.

Practical recommendations: In the comprehensive treatment strategy for patients with CHF developed against the background of mitral valve disease, along with hemodynamic

correction, continuous monitoring (EchoCG, ECG, NT-proBNP) and therapy (anticoagulants, antiarrhythmics) targeting left atrial remodeling and its arrhythmogenic consequences should be implemented. Furthermore, performing surgical intervention before the development of irreversible changes in the left atrium may be an important factor in the prevention of atrial fibrillation.

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References:

1.Watkins, D. A., Johnson, C. O., Colquhoun, S. M., et al. Global, regional, and national burden of rheumatic heart disease, 1990–2015. The New England Journal of Medicine. 2017;377(8):713–722.

2.Bursi, F., Enriquez-Sarano, M., Nkomo, V. T., et al. Heart failure after myocardial infarction: A review. European Heart Journal. 2007;28(23):2866–2872.

3.Koelling, T. M., Aaronson, K. D., Cody, R. J., et al. Prognostic significance of mitral regurgitation and tricuspid regurgitation in patients with left ventricular systolic dysfunction. American Heart Journal. 2002;144(3):524–529.

4.Agricola, E., Oppizzi, M., Pisani, M., et al. Ischemic mitral regurgitation: Mechanisms and echocardiographic classification. European Journal of Echocardiography. 2008;9(2):207–221.

5.Mirabel, M., Lung, B., Baron, G., et al. What are the characteristics of patients with severe, symptomatic, mitral regurgitation who are denied surgery? European Heart Journal. 2007;28(11):1358–1365.

6.Guazzi, M., & Borlaug, B. A. Pulmonary hypertension due to left heart disease. Circulation. 2012;126(8):975–990.

7.Negi, P. C., Sondhi, S., Rana, V., et al. Prevalence, risk factors and outcomes of atrial fibrillation in rheumatic heart disease: The HP-RF/RHD Registry. Indian Heart Journal. 2018;70(1):68–73.

8.Hugenholtz, P. G., Ryan, T. J., Stein, S. W., & Abelmann, W. H. The spectrum of pure mitral stenosis. Hemodynamic studies in relation to clinical disability. The American Journal of Cardiology. 1962;10:773–784.

9.January, C. T., Wann, L. S., Alpert, J. S., et al. 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation. Circulation. 2014;130(23):2071–2104.

10.Obadia, J. F., el Farra, M., Bastien, O. H., et al. Outcome of atrial fibrillation after mitral valve repair. The Journal of Thoracic and Cardiovascular Surgery. 1997;114(2):179–185.

11.Bozkurt, B. 2021 ACC/AHA key data elements and definitions for heart failure. Journal of the American College of Cardiology. 2021;77(16):2053–2150.

12.McDonagh, T. A., Metra, M., Adamo, M., et al. 2021 ESC guidelines for the diagnosis and treatment of acute and chronic heart failure. European Heart Journal. 2021;42(36):3599–3726.

13.Mueller, C., McDonald, K., de Boer, R. A., et al. Heart Failure Association of the European Society of Cardiology practical guidance on the use of natriuretic peptide concentrations. European Journal of Heart Failure. 2019;21(6):715–731.

14.Maisel, A., Hollander, J. E., Guss, D., et al. Primary results of the Rapid Emergency Department Heart Failure Outpatient Trial (REDHOT). Journal of the American College of Cardiology. 2004;44(6):1328–1333.

15.Roitberg, G. E., & Strutynsky, A. V. Internal diseases. Cardiovascular system. MEDpress-inform. 2014.
16.Rudiktiyo, E., et al. Left Ventricle Myocardial Work Correlated with Functional Capacity in Severe Rheumatic Mitral Stenosis with Preserved Left Ventricular Ejection Fraction. 2024.