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From Diagnosis to Intervention: The Role of Echocardiography in Guiding Management of a Giant Left Atrium in Rheumatic Mitral Stenosis

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Abstract

Giant left atrium (GLA) is an uncommon but notable manifestation of advanced rheumatic heart disease (RHD), particularly in patients with longstanding rheumatic mitral stenosis. Extreme atrial remodeling may be associated with intracavitary blood stasis, pulmonary hypertension, and progressive right-sided cardiac involvement. A 50-year-old woman with congestive heart failure secondary to RHD presented with intermittent dyspnea, easy fatigability, and palpitations. Electrocardiography revealed atrial fibrillation (AF), and chest radiography demonstrated cardiomegaly with pulmonary vascular congestion. Echocardiography showed severe rheumatic mitral stenosis (MS) with mild mitral regurgitation and a Wilkins score of 9. The left atrium was markedly enlarged, measuring 7.0 cm, with a left atrial volume index (LAVI) of 239 mL/m² and spontaneous echo contrast without identifiable thrombus. Additional findings included mild aortic regurgitation, moderate tricuspid regurgitation, mild pulmonary regurgitation, a D-shaped left ventricle during systole and diastole, isolated post-capillary pulmonary hypertension, and reduced right ventricular systolic function despite preserved left ventricular systolic function. This case demonstrated the progression of chronic rheumatic mitral obstruction from sustained left atrial pressure overload to extreme atrial remodeling, blood stasis, pulmonary hypertension, and right ventricular involvement. Preserved left ventricular systolic function may coexist with substantial advanced hemodynamic impairment. Mitral valve replacement with left atrial reduction and left atrial appendage occlusion was recommended. This case highlights the central role of comprehensive echocardiography in identifying the structural, hemodynamic, and thromboembolic consequences of severe rheumatic MS with GLA. Echocardiography provided essential information for individualized therapeutic decision-making.

INTRODUCTION

Rheumatic heart disease (RHD) remains an important cause of preventable cardiovascular morbidity and mortality, with a disproportionate burden in low- and middle-income countries despite substantial reductions in disease-related mortality over recent decades. (Ghamari et al., 2022; Zühlke et al., 2026) RHD represents the chronic valvular consequence of acute rheumatic fever and may progress silently over years before clinically significant valvular dysfunction becomes apparent. The mitral valve is

particularly susceptible to rheumatic injury, and progressive leaflet thickening, commissural fusion, and subvalvular involvement can ultimately result in clinically significant mitral stenosis. In advanced disease, the consequences extend beyond the valve itself, involving the left atrium, pulmonary circulation, and eventually the right side of the heart (Figueiredo et al., 2024; Soesanto et al., 2025).

Rheumatic mitral stenosis causes chronic obstruction to left atrial emptying, resulting in sustained elevation of left atrial pressure and progressive atrial remodeling. As the disease advances, elevated left atrial pressure may be transmitted backward to the pulmonary circulation, contributing to pulmonary hypertension and subsequent right-sided hemodynamic consequences. (Otto et al., 2021; Vahanian et al., 2022) Left atrial enlargement is therefore an important structural manifestation of the chronic pressure burden associated with mitral stenosis rather than merely an incidental echocardiographic finding. Current valvular heart disease guidelines emphasize assessment not only of mitral valve anatomy and stenosis severity but also of downstream consequences, including left atrial enlargement, pulmonary pressures, concomitant valvular lesions, and right ventricular involvement when determining disease severity and appropriate management strategies for rheumatic mitral stenosis (Kim, 2023; Niu et al., 2022; Praz et al., 2025).

At the extreme end of this remodeling spectrum is giant left atrium (GLA), an uncommon manifestation that remains closely associated with longstanding rheumatic mitral valve disease.(Bhasin et al., 2024; Med Sidi El Moctar et al., 2023; Pandit et al., 2021) Although different criteria have been used to define GLA, an anteroposterior left atrial diameter exceeding approximately 65 mm has frequently been applied in contemporary literature. Progressive enlargement may have important clinical implications beyond chamber size alone. Marked atrial dilatation promotes low-flow conditions and blood stasis, creating a substrate for spontaneous echo contrast, atrial thrombus formation, and systemic thromboembolism. Dense spontaneous echo contrast in the left atrium is recognized as a marker of increased thromboembolic risk in patients with rheumatic mitral stenosis. In more advanced cases, the enlarged atrium may distort or compress adjacent cardiac and extracardiac structures, while longstanding pulmonary vascular consequences may accompany progressive right-sided cardiac involvement (Baumgartner et al., 2009; Gewitz et al., 2015; Watkins et al., 2017).

The urgency of this research is underscored by the continuing burden of RHD in Indonesia and other developing countries, where advanced presentations remain common. A multicenter Indonesian study involving 3,431 patients across 21 hospitals reported a high prevalence of dyspnea, mitral stenosis, atrial fibrillation, pulmonary hypertension, and right heart involvement (Soesanto et al., 2025). This case report provides novelty by illustrating the progression of chronic rheumatic mitral obstruction from sustained left atrial pressure overload to extreme atrial remodeling, blood stasis, pulmonary hypertension, and right ventricular involvement, a phenotype that has become increasingly uncommon in high-income countries but remains encountered in Indonesia. The purpose of this report is to highlight the central role of comprehensive

echocardiography in identifying the structural, hemodynamic, and thromboembolic consequences of severe rheumatic mitral stenosis with GLA and to demonstrate how these findings guide individualized therapeutic decision-making (Lang et al., 2015; Remenyi et al., 2013) .

The presence of GLA may also influence the therapeutic approach to advanced rheumatic mitral valve disease. Definitive correction of the underlying mitral valve lesion remains central to management when intervention is indicated, while optimal treatment strategies for the markedly enlarged atrium itself remain less standardized. Surgical left atrial reduction performed concomitantly with mitral valve surgery has been described as a strategy to restore more physiological atrial dimensions and address the consequences of extreme atrial enlargement. (Adalti et al., 2018; Ríos-Ortega et al., 2023; Suyanto, 2016) Observational surgical series have demonstrated substantial postoperative reductions in left atrial dimensions and reported favorable functional and clinical outcomes, although available evidence remains limited and patient selection continues to require individualized consideration.

Giant left atrium has become increasingly uncommon in the contemporary era of earlier echocardiographic detection and intervention, making advanced presentations particularly valuable for understanding the natural structural consequences of chronic rheumatic valve disease. This report presents a case of GLA associated with severe rheumatic mitral stenosis and mild mitral regurgitation, accompanied by spontaneous echo contrast, pulmonary hypertension, right-sided cardiac involvement, and reduced right ventricular systolic function despite preserved left ventricular systolic function. The present case highlights the role of comprehensive echocardiography in characterizing GLA with severe rheumatic mitral stenosis and spontaneous echo contrast by integrating structural findings, progressive cardiopulmonary hemodynamic consequences, and thromboembolic risk indicators to guide therapeutic decision-making.

The objective of this case report was to describe a case of GLA associated with severe rheumatic mitral stenosis and mild mitral regurgitation, accompanied by spontaneous echo contrast, pulmonary hypertension, right-sided cardiac involvement, and reduced right ventricular systolic function despite preserved left ventricular systolic function, and to demonstrate the role of comprehensive echocardiography in guiding management. The contribution of this report is twofold. Clinically, it provides detailed echocardiographic characterization of advanced rheumatic mitral valve disease with GLA, illustrating the integration of structural, hemodynamic, and thromboembolic findings for therapeutic decision-making. Educationally, it reinforces the importance of evaluating atrial remodeling, blood stasis, pulmonary hemodynamics, and ventricular function beyond valve severity alone in patients with advanced rheumatic mitral stenosis. The benefits of this report include increasing clinician awareness regarding comprehensive echocardiographic assessment for individualized GLA management and supporting evidence-based decisions regarding concomitant left atrial reduction and left atrial appendage (LAA) occlusion during mitral valve surgery.

METHOD

This report described a single clinical case managed at the Department of Cardiology and Vascular Medicine, Bali Mandara Regional General Hospital, Denpasar, Bali, Indonesia. Data were obtained through direct clinical examination, review of the patient's medical records, and diagnostic investigations, including electrocardiography, chest radiography, and comprehensive transthoracic echocardiography. Echocardiographic assessment was performed according to current recommendations for evaluating rheumatic mitral valve disease, including assessment of mitral stenosis severity using the Wilkins score, measurement of left atrial size and left atrial volume index (LAVI), evaluation of spontaneous echo contrast, and assessment of pulmonary artery pressure and right ventricular systolic function. Relevant literature was reviewed to support the interpretation of clinical and echocardiographic findings. Written informed consent was obtained from the patient for the use of clinical data and imaging findings for academic and publication purposes. Patient confidentiality was maintained throughout the reporting process in accordance with the CARE (CAse REport) guidelines.

RESULTS AND DISCUSSION

A 50-year-old woman with congestive heart failure secondary to rheumatic heart disease presented with intermittent exertional dyspnea, easy fatigability, and recurrent palpitations. Vital signs were stable (BP 119/80 mmHg, HR 85 bpm, RR 20 bpm, SpO2 99%). Examination revealed an irregularly irregular rhythm with a low-pitched diastolic murmur at the apex, without jugular venous distension or peripheral edema. ECG showed atrial fibrillation, and chest X-ray showed cardiomegaly with pulmonary vascular congestion. The patient was on warfarin, bisoprolol, candesartan, spironolactone, and furosemide with serial INR monitoring.

Transthoracic echocardiography revealed severe mitral stenosis with preserved LV systolic function (LVEF 51.3%) but a giant left atrium (diameter 70 mm; LAVI 239 mL/m^2), with spontaneous echo contrast (SEC) but no thrombus. Given the combination of atrial fibrillation, severe mitral stenosis, giant left atrium, and SEC, transesophageal echocardiography was performed and confirmed these findings, along with leaflet thickening, commissural fusion, and restricted posterior leaflet motion (mitral valve area 0.44 cm^2 by planimetry; Wilkins score 9; revised echocardiographic score 11 — unfavorable for percutaneous mitral commissurotomy). Moderate tricuspid regurgitation, reduced right ventricular systolic function (TAPSE 13.6 mm), a D-shaped left ventricle, and hemodynamic evidence of pulmonary hypertension (mean PAP 29 mmHg) were also found. Based on this comprehensive assessment, mitral valve replacement with concomitant left atrial reduction and left atrial appendage occlusion was recommended as the definitive treatment strategy.

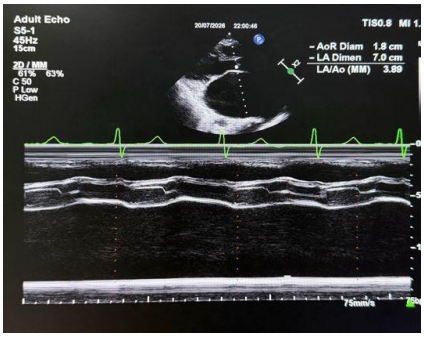

Figure 1. M-mode echocardiographic assessment demonstrating marked left atrial enlargement, with a left atrial diameter of 7.0 cm.

Figure 2. Two-dimensional echocardiographic assessment demonstrating severe left atrial enlargement, with a left atrial volume index of 239.8 mL/m².

Figure 3. Two-dimensional echocardiographic planimetry demonstrating a severely reduced mitral valve area of 0.45 cm².

Figure 4. Continuous-wave Doppler assessment demonstrating an elevated mean transmitral gradient of 12 mmHg, with mitral valve area measurements of 0.37 cm² by the velocity-time integral method and 0.50 cm² by pressure half-time.

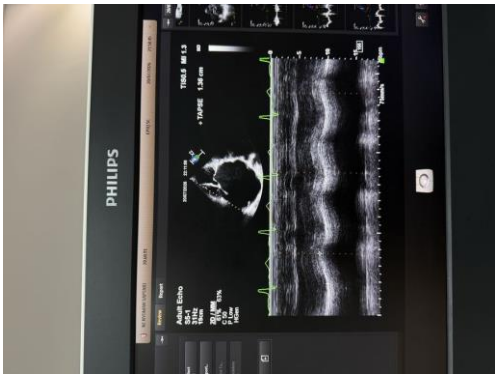

Figure 5. M-mode assessment of right ventricular systolic function demonstrating reduced tricuspid annular plane systolic excursion (TAPSE) of 13.6 mm.

Discussion

This case illustrates an advanced manifestation of rheumatic heart disease (RHD), characterized by severe rheumatic mitral stenosis with giant left atrial (GLA) remodeling. Although RHD remains a substantial global cardiovascular burden, advanced structural manifestations are increasingly less common where valvular disease is recognized and treated earlier.1,2 The coexistence of marked LA enlargement, spontaneous echo contrast, pulmonary hypertension, right atrial dilatation, and reduced RV systolic function demonstrates progression from chronic rheumatic valve disease to complex cardiopulmonary involvement.3,4 Contemporary Indonesian data similarly indicate that mitral stenosis remains the predominant rheumatic valve lesion, with advanced disease frequently associated with pulmonary hypertension and right-heart involvement.11

The most distinctive feature of this case was the giant left atrium. Although no universally accepted definition exists, an anteroposterior left atrial diameter >65 mm is commonly used, and the 7.0-cm diameter in this patient fulfills this criterion.5,7 In rheumatic mitral stenosis, chronic obstruction from commissural fusion and leaflet/subvalvular involvement leads to persistent elevation of left atrial pressure and progressive atrial remodeling.3,4 GLA represents more than an anatomical enlargement: in this patient it was accompanied by SEC, pulmonary hypertension, right atrial dilatation, and impaired RV systolic function, reflecting advanced hemodynamic consequences and an important marker of disease progression.12 The presence of SEC further indicates significant blood stasis and thromboembolic risk, and current valvular guidelines recognize dense LA SEC as a feature associated with increased thromboembolic risk in rheumatic mitral stenosis.13 Although atrial fibrillation was not explicitly documented as the indication, the patient was appropriately maintained on warfarin with serial INR monitoring.

The downstream pulmonary and right-sided consequences further emphasize the advanced hemodynamic burden of rheumatic mitral stenosis, as elevated left atrial pressure is transmitted backward into the pulmonary circulation, increasing right ventricular afterload and potentially leading to right-sided remodeling and dysfunction.3,4 In this patient, isolated post-capillary pulmonary hypertension was

accompanied by moderate tricuspid regurgitation, right atrial dilatation, a D-shaped left ventricle, and reduced RV systolic function despite preserved LV systolic function. Pulmonary hypertension in rheumatic mitral stenosis has been associated with poorer outcomes after mitral valve replacement, while impaired RV–pulmonary artery coupling may further predict early mortality and perioperative complications.14,15 Although RV–pulmonary artery coupling was not assessed in this case, the reduced RV systolic function underscores the importance of evaluating pulmonary hemodynamics and RV performance in advanced disease.

The therapeutic strategy was guided by the severity of mitral stenosis and its extensive structural and hemodynamic consequences. With a Wilkins score of 9, the valve anatomy was unfavorable for percutaneous mitral commissurotomy according to the 2025 ESC/EACTS guidelines.13 Surgical management — mitral valve replacement with concomitant left atrial reduction and left atrial appendage occlusion — was therefore recommended to address both the primary valve lesion and the extensive atrial remodeling. Concomitant LA reduction may be considered in selected patients with GLA, as mitral valve replacement alone may not immediately reverse severe atrial remodeling; previous surgical series have shown substantial reductions in LA diameter and volume with combined surgery, although these studies remain observational.8–10 No LA or LAA thrombus was documented, so LAA occlusion was proposed as a preventive part of the surgical strategy rather than treatment of an established thrombus.

Notably, LV systolic function was preserved despite extensive pulmonary and right-sided involvement, since severe rheumatic mitral stenosis primarily causes LV inflow obstruction and LV systolic function may remain preserved even with significant pulmonary hypertension and RV dysfunction.3,4,14,15 This underscores that disease severity cannot be assessed by LV systolic function alone, and reflects the continuing clinical relevance of advanced RHD in contemporary practice, as a multicentre Indonesian study of 3,431 patients across 21 hospitals reported a high prevalence of dyspnea, mitral stenosis, atrial fibrillation, pulmonary hypertension, and right-heart involvement.11 Overall, the documented findings a 7.0-cm LA, SEC, pulmonary hypertension, right atrial dilatation, moderate TR, and reduced RV systolic function despite preserved LV function objectively demonstrate an advanced phenotype of rheumatic mitral stenosis and supported an integrated surgical strategy of mitral valve replacement with concomitant LA reduction and LAA occlusion, although postoperative outcomes were not available in this case.

CONCLUSION

GLA represents an advanced structural manifestation of rheumatic mitral stenosis, reflecting the cumulative effects of chronic left atrial pressure overload and progressive cardiac remodeling. In this case, comprehensive echocardiographic assessment identified severe mitral stenosis, extensive left atrial enlargement with spontaneous echo contrast, and significant right-sided cardiac and pulmonary consequences despite preserved left ventricular systolic function. These findings were essential for defining disease severity,

evaluating suitability for percutaneous intervention, and supporting the decision for surgical management. This case emphasizes that echocardiography should extend beyond the assessment of valve severity to include comprehensive evaluation of atrial remodeling, blood stasis, pulmonary hemodynamics, and ventricular function to guide individualized management of advanced rheumatic mitral valve disease.

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